The New England
Journal of Medicine

VOLUME 207 SEPTEMBER 22, 1932 NUMBER 12

NEW ENGLAND HEART ASSOCIATION

THE ANATOMY AND PHYSIOLOGY OF CONGENITAL CARDIOVASCULAR DISEASE*

BY A. CARLTON ERNSTENE, M.D.†

ABBOTT has classified congenital defects of the cardiovascular system in three principal groups: (1) the a-cyanotic group, (2) the group of cases having an arteriovenous shunt with possible transient cyanosis due to reversal of flow through the defect, and (3) the cyanotic group (morbus caeruleus).

The A-cyanotic Group. In this group there is no abnormal communication between the pulmonary and systemic circulations and, therefore, no cause for congenital cyanosis. The most important member of the group is coarctation of the aorta of the adult type. This consists of a localized stenosis or atresia of the descending arch of the aorta at the point of insertion of the ductus arteriosus. The aorta above the level of the stenosis may be dilated and thin and at times is the site of a dissecting aneurysm. The ductus arteriosus generally is partly or completely closed but occasionally remains patent. Cardiac enlargement is usually but not always present. One of the most striking features of the condition is the development of collateral arterial circulation between the branches of the aorta above and below the site of stenosis. This is accomplished mainly by the following routes: (1) by anastomoses between the superior intercostal artery of the subclavian and the first aortic intercostal arising from the aorta just below the site of the constriction, (2) by anastomoses between the aortic intercostal and the subscapular artery and its branches, particularly the circumflex scapulae, and (3) by anastomoses between the internal mammary and deep epigastric arteries. The extent to which each of these vessels takes part in the collateral circulation varies considerably in different patients. The collateral circulation may manifest itself clinically by the presence of pulsating vessels over the back, epigastrium and lower anterior part of the thorax, prominent subclavian and subscapular arteries and palpable deep epigastric arteries. In addition, roentgenographic examination of the thorax reveals erosions of the inferior borders of the ribs bilaterally.

Because of the resistance offered to the flow of blood by the stenosed aorta and the collateral arterial pathways, patients with coarctation of the aorta present hypertension in the vessels of the arms and relatively low blood pressure in the legs. In addition, the femoral pulse wave often is retarded and diminished in amplitude or absent. Because of the importance of this diagnostic sign, the femoral pulse should be palpated in all individuals with elevated brachial blood pressure. The height of the brachial blood pressure and the diminution and retardation of the femoral pulse wave depend on the degree of stenosis of the aorta and the calibre and directness of route of the collateral pathways.

Measurements of the velocity of arterial blood flow by the radioactive method in one patient with coarctation of the aorta revealed a prolongation of the time necessary for the radioactive material to traverse the distance between the heart and the femoral artery. This indicated the circuitous route traveled by the blood before it reached the lower extremities and the increased resistance it encountered in coursing through the collateral channels. There was also prolongation of the time necessary for the blood to flow from the femoral artery to the superficial vessels of the foot, due probably to the low gradient of pressure in the larger vessels of the legs.

A second member of the a-cyanotic group of cases is subaortic stenosis, a condition in which the cavity of the left ventricle is encroached upon by a narrow zone of thickening of the endocardium a short distance below the aortic valve. The defect is commonly attended by hypertrophy of the left ventricle.

Idiopathic congenital cardiac hypertrophy is another example of the a-cyanotic type of congenital heart disease. In this condition an abnormally large heart is present at birth, the hypertrophy usually involving the left ventricle more than the right. The heart valves are normal. Microscopic examination of the myocardium reveals degeneration of the muscle fibres with increased fibrous tissue. The cause of the hypertrophy is unknown, but it is of interest that in a few of the recorded cases the left coronary artery had its origin from the pulmonary artery.

Defects Giving Rise to Arteriovenous Shunts with Possible Transient or Terminal Reversal of

*Read at the meeting of the New England Heart Association, Cardiac Course, at Boston, December 2, 1931.

†Ernstene—Assistant in Medicine, Beth Israel Hospital. For record and address of author see "This Week's Issue," page 553.

Flow. This group is composed of localized defects of the interauricular, interventricular and aortic septa and uncomplicated patency of the ductus arteriosus. Individual defects of this kind are much less common than are similar defects combined with other congenital cardiovascular anomalies. The uncomplicated defects are accompanied by a leakage of blood from the arterial to the venous side of the circulation. The direction of the leakage is due, of course, to the fact that the pressure in the left chambers of the heart and in the aorta normally is higher than in the corresponding part of the pulmonary circulation. As long as the direction of the shunt through localized defects of the cardiac septa is from the arterial to the venous side of the circulation, no cause for congenital cyanosis exists. At anytime, however, an increase in pressure in the pulmonary circulation resulting either from pathologic processes in the lungs or from a failing left ventricle may convert the arterial-venous shunt into a venous-arterial one. If the venous-arterial shunt attains sufficient magnitude, cyanosis develops (cyanose tardive).

Uncomplicated patency of the ductus arteriosus is the most common defect in this type of congenital cardiovascular disease. In this condition, the leakage of blood from the aorta to the pulmonary artery increases the work of both ventricles and causes hypertrophy and dilatation of both chambers. Dilatation of the pulmonary artery also develops.

Patency of the foramen ovale is, as a rule, of little or no clinical importance. Usually, however, the increased volume of blood in the right auricle and ventricle produces some enlargement of these chambers. At times the pulmonary artery is dilated and the aorta hypoplastic.

Defects of the interventricular septum are situated in practically all cases at the base of the septum directly beneath the aortic valve in the region of the so-called undefended space. Uncomplicated defects of this kind seem to place very little added burden on the right ventricle and to interfere in no way with normal cardiac function. The pulmonary artery usually is not dilated and the heart often is of normal size. In many instances, a tendinous patch of fibrosis is found on the wall of the right ventricle opposite the septal defect. Terminal or transient cyanosis, due to conversion of the arterial-venous shunt into a venous-arterial one, is much less common than in defects of the auricular septum or patent ductus arteriosus.

Cyanotic Group. In this group are included all those congenital cardiac defects accompanied by permanent cyanosis. The degree of cyanosis varies from moderate to extreme depending on the type of lesion and the associated cardiovascular anomalies. In almost all instances, a combination of two or more cardiac defects is found at necropsy.

The most frequent cause of congenital cyanosis in adult life and the most frequent of all combinations of congenital cardiac defects is the tetralogy of Fallot, consisting of a defect of the ventricular septum, pulmonary or infundibular stenosis, dextroposition of the aorta and hypertrophy of the right ventricle. The aorta arises from both ventricles above a large, smooth-walled defect at the upper part of the interventricular septum.

Patients with the tetralogy of Fallot present all the significant features of the pathologic physiology of the cyanotic type of congenital heart disease. (1) They have a high degree of cyanosis, due principally to the fact that the dextro-posed aorta receives blood from both right and left ventricles and in part to stasis of blood in the dilated capillaries of the systemic circulation. In order that cyanosis may result from a venous-arterial shunt, at least one-third of the total blood volume must pass through the shunt. Actually it has been calculated that as much as 70% of the total blood volume may pass through the shunt in patients with the tetralogy of Fallot.

(2) Clubbing of the fingers and toes usually is present.

(3) In the earlier stages, the patient may have slight or no dyspnea at rest even though there is definite cyanosis. Muscular exertion may precipitate a paroxysm of dyspnea due apparently to an increase in the magnitude of the venous shunt which also increases the degree of cyanosis.

(4) In the later stages, examination of the blood commonly reveals an increase in the number of red blood cells. This represents an attempt on the part of the hematopoietic system to compensate for the deficient oxygenation of the tissues. The red blood cell count may be as high as twelve million per cubic millimeter. There is an accompanying increase in the total blood volume which accounts for the plethoric appearance of the patients. The viscosity of the blood also is increased.

REFERENCES

Abbott, M. E., and Weiss, E.: The Diagnosis of Congenital Cardiac Disease. Blumer's Bedside Diagnosis, W. B. Saunders Co., Philadelphia, 1928, 2, 353.

Blumgart, H. L., Lawrence, J. S., and Ernstene, A. C.: The Dynamics of the Circulation in Coarctation (Stenosis of the Isthmus) of the Aorta of the Adult Type. Arch. Int. Med., 1931, 47, 806.

Ernstene, A. C., and Robins, S. A.: The Roentgenologic Diagnosis of Stenosis of the Descending Arch (Coarctation) of the Aorta. Am. J. Roentgenol., 1931, 25, 243.

White, P. D.: Heart Disease. Macmillan Co., New York, 1931.

CONGENITAL HEART DISEASE*

Clinical Types and Diagnosis

BY HOWARD B. SPRAGUE, M.D.†

THERE has been a notable lack of interest in the subject of congenital heart disease by the general practitioner. It is true that it represents only about two per cent of the total cases of organic heart disease in this part of the country and about five per cent of the heart disease of childhood.

Two arguments are commonly raised against an attempt to make a differential diagnosis of the various lesions in congenital anomalies of the heart. The first is that the diagnosis is extremely obscure and the second is that there is no specific treatment for the condition.

The first objection would certainly be supported by the great number of complicated possibilities outlined in the survey by Dr. Maude Abbott. She reports eight hundred and fifty cases studied pathologically and divides the lesions into a total of sixty-four sub-groups. This makes the combined occurrence of a single lesion appearing as the chief defect plus its combination with other lesions as one thousand nine hundred and sixty-eight. One must, however, realize that this survey was made of cases studied at autopsy and not of those diagnosed clinically. As a matter of fact, many of these lesions could not have been diagnosed at the bedside and there are nineteen sub-groups in which the mean duration of life was under two years. These include infants dying at birth and during fetal life, and represent pathological curiosities in many cases.

As regards the second objection it must be admitted that the therapy consists in protecting these patients from the effects of strain and infection, but it is by an attempt to make a differential diagnosis that we can learn more of the problems of the transmission of the defect and can give an intelligent prognosis.

The cases which I shall describe are limited to those that survive beyond the age of two years and, except for two rare types, the average duration of life is over eleven years. The point of view of a physician seeing older children and adults, must of necessity be different from that of the pediatrician who is often called upon to make a diagnosis of congenital heart defect during the first year of life. At this time the diagnosis is far more difficult as the characteristic murmurs frequently do not appear during the first year. However, it is sometimes thought that the graver abnormalities of cardiovascular structure are not consistent with life beyond a few days or months and it is, therefore, interesting to remember that a patient with only two heart chambers, that is, with a heart resembling that of a fish, has been able to live to the age of sixteen years and that one with three chambers, the amphibian heart with two auricles and one ventricle, has survived to the age of thirty-five years.

Before describing the types of defect which can, with some certainty, be diagnosed clinically, I should like to say something about the diagnosis of patent foramen ovale. It is very common for doctors who have not had special training to consider as the most probable diagnosis in any case of congenital heart disease, that of patency of the foramen ovale. This is due to the obstetrical teaching concerning the blue baby. As a matter of fact, patent foramen ovale of an important degree is an extremely rare anomaly of the heart and should not be considered as a likely cause of cyanosis in this disease. Neither Dr. Paul White nor I has ever made the diagnosis clinically. In Dr. Abbott's series there are a few cases of wide defects in the auricular septum but patent foramen ovale as a primary lesion occurred only thirty-two times (3.7%) and the mean duration of life was thirty-two and one-half years. In two hundred and twenty-six cases it complicated other lesions.

I wish to discuss eight types of congenital heart disease which can usually be diagnosed. Referring to Abbott's figures once more we find that the cases in these groups represent forty-five per cent of her eight hundred and fifty cases. Each type has a characteristic sign or collection of signs.

1. ECTOPIA CORDIS

The first group can be rapidly dismissed, but is included because it is an anomaly of the whole heart and readily diagnosable. The heart in ectopia cordis may be in the abdominal cavity or outside the chest wall. It is frequently associated with a midline defect of the entire anterior abdominal wall. I have seen one such case operated on but as is usual there was insufficient lateral tissue to allow a reposition of the organs. It is not commonly known that the maximum duration of life has been noted as fifteen months and the mean as one month.

2. CONGENITAL IDIOPATHIC HYPERTROPHY

The diagnosis of this condition is made by exclusion. The maximum reported duration of life is four years and the mean, sixteen months.

*Read at the meeting of the New England Heart Association, Cardiac Course, at Boston, December 2, 1931.

†Sprague—Visiting Physician and Research Director, House of the Good Samaritan. For record and address of author see "This Week's Issue," page 553.

The diagnosis, therefore, is made by finding a generally enlarged heart in a young child in which there is no evidence of pericardial effusion or valvular disease. There may be a slight general systolic murmur. There is usually slight, if any cyanosis, except as a terminal sign.

3. DEXTROCARDIA

This diagnosis can be made readily if one is on the lookout for it, particularly, when there is the true situs inversus. The condition can be proved by means of the electrocardiogram. It shows a complete inversion of the waves in lead one and a transposition of leads two and three. The mean duration of life in Abbott's series is thirteen years and the maximum, forty-six. It is likely, however, that older cases exist because when uncomplicated the anomaly is well tolerated.

4. INTERVENTRICULAR SEPTAL DEFECT (ROGER'S DISEASE)

The usual defect in the septum between the ventricles is situated high up in the fibrous portion of the septum. The excess of pressure in the left ventricle over that of the right during the cardiac cycle results in a flow of blood from left to right so that such patients are not cyanotic except in the terminal stages when there may be a reversal of flow, particularly if conditions are present which produce obstruction to the pulmonary circulation. The heart may or may not be enlarged, the prognosis being somewhat determined by the evidence of enlargement. The diagnostic signs consist of a rough systolic murmur and thrill in the midsternal region or at the third and fourth intercostal spaces near the sternum. The electrocardiogram frequently shows some degree of right axis deviation. The oldest reported case in Abbott's series studied at necropsy was forty-four years of age and the average thirteen and three-quarters years. In rare instances the defect in the septum interferes with the development of the conduction system as it passes through the septum. The result is to produce high-grade or complete dissociation between auricles and ventricles.

I have, at present, under observation, a girl of seventeen with this combination of lesions. She has been seen by Dr. White and me for the past seven and one-half years and is symptom-free and able to engage in mild athletics.

5. PATENT DUCTUS ARTERIOSUS

Milder degrees of patency of the ductus arteriosus are common. This was found in 96 cases in five thousand autopsies at the Massachusetts General Hospital. It occurred in ninety-seven cases in Abbott's series. As the pressure in the aorta is usually greater than that in the pulmonary artery the common finding is an arteriovenous shunt. The patients are, therefore, not cyanotic. The diagnosis is made by finding a continuous murmur and thrill maximal at the second left interspace near the sternum. In addition there is usually enlargement of the pulmonary artery on physical examination and by x-ray due to the increased pressure from the arterial blood flowing into it. The heart may be generally enlarged because there is a certain strain on both ventricles. However, if the amount of blood which flows into the pulmonary artery is not great, there may be very little evidence of enlargement of the heart. Marked degrees of regurgitation from the aorta are not necessarily associated with loud murmurs, the reverse may be true. In exaggerated cases the findings in the peripheral arteries resemble those in marked aortic regurgitation, such as a Corrigan pulse and Duroziez's sign. In rare cases the murmur may be only systolic in time and in other cases heard loudest at the apex. In those with slight murmurs at the base it is well to rule out the possibility of their origin from venous hums above the clavicle. The latter are common normal occurrences in children. The oldest case of patent ductus arteriosus that lived to the age of sixty-six was studied by Dr. Paul D. White. The mean duration of life is thirty-three and one-quarter years.

6. SUBAORTIC AND AORTIC STENOSIS

It is very unusual to find rheumatic aortic stenosis in young children and discovery of the characteristic systolic murmur and thrill at the second right interspace in an infant or young child, suggests very strongly the diagnosis of congenital stenosis of the conus of the left ventricle or the aortic valve. The oldest reported case was fifty-eight years of age and the average twenty-two and two-thirds years in Abbott's series. The differential diagnosis from acquired aortic stenosis in later life is difficult.

7. TETRALOGY OF FALLOT

This combination of lesions is by far the commonest cause of cyanosis in congenital heart patients surviving beyond the first few months of life. The oldest case is one that died at the age of fifty-nine and two-thirds years and was observed by Dr. White and me. The average duration of life is eleven and one-half years. The tetralogy consists of stenosis of the infundibulum of the right ventricle and of the pulmonary valve, defect in the interventricular septum, dextroposition of the aorta, and enlargement of the right ventricle. The obstruction to the outflow of blood from the right ventricle through the pulmonary artery results in an increased pressure in the right ventricle. This causes the blood to flow through the septal defect and to be partly discharged through the aorta which usually lies across the defect. The natural re-

sult of this is marked enlargement of the right ventricle as shown by physical examination, x-ray and electrocardiogram, and cyanosis with clubbing of the fingers and toes. The heart may show a rough systolic murmur and thrill loudest at the second left interspace near the sternum but in cases in which the conus pulmonalis is markedly stenotic the murmur may be heard farther down, more mesially, and much less distinctly.

8. COARCTATION OF THE AORTA (ADULT TYPE)

During the development of the ductus arteriosus there may be a narrowing in the descending aorta at the point where the ductus leaves it. This narrowing may be of sufficient degree to cause marked circulatory changes. As might be expected, the heart tends to hypertrophy when forced to contract against this obstruction. There is a hypertension of the upper part of the body and a low blood pressure in the arterial system below the coarctation. This produces a paradoxical blood pressure condition in the arms and legs, the pressure in the legs being impossible to record in some cases. A compensatory circulation is set up by anastomoses of the internal mammary and epigastric arteries, and through the dilated intercostal vessels. The latter with their branches may at times be seen as strongly pulsating vessels over the chest wall, particularly at the back, and there results a pressure atrophy of the lower margins of the ribs producing a characteristic scalloping which can be seen in the x-ray plate. The x-ray shows in addition a small aortic knob. There is often a long systolic murmur heard best over the upper part of the back. The condition has been observed up to the age of ninety-two years and the average duration of life is thirty-three and one-half years.

CONCLUSION

At the present time it is wiser, I believe, to restrict our attempts to the differential diagnosis of the anomalies in congenital heart disease to the groups which I have outlined. It is usually only confusing to think of all the possible combinations of congenital heart defect. If one can be sure of diagnosing these types, it may lead later to a better differentiation of the more obscure lesions which have not a characteristic combination of clinical signs.

A paper entitled "The Incidence and Prognosis of Congenital Heart Disease" by Hyman Green, M.D. and Paul Emerson, M.D. was read by Dr. Green. Studies on the subject are not yet completed. The paper will be published in a later issue.

DISCUSSION

DR. KENNETH BLACKFAN: I have been very much interested in the papers which have been presented this afternoon even though the points which have been brought out regarding the subject at hand have somewhat decreased the optimism I have felt in regard to the possibility of recognizing definitely a larger number of defects of the heart. One of the reasons which, I believe, make the diagnosis of congenital heart diseases so difficult is the fact that most of our knowledge has come from descriptions of the defects at autopsy. I am inclined to hope that with the new methods of approach to the subject such as the roentgen ray and the electrocardiograph together with the correlation of clinical and physical findings and together with these embryological studies of the heart at autopsy, one may remain optimistic and look forward to finding clinical evidences by which we can recognize a larger number of anomalies than those emphasized by Dr. Sprague. The importance of diagnosis is obvious for upon it rest more accurate prognosis and sound treatment. Even if no curative treatment can be suggested, the advice to an anxious parent that the heart is fully compensating and that no treatment is indicated, is as helpful as prescribing a drug. That new information in respect to prognosis has been found along the lines suggested, i.e., following patients over a long period of time is shown clearly by the studies reported by Dr. Green and Dr. Emerson.

Now if we consider the underlying causes of congenital cardiac defects we find that three general theories have been advanced, (1) that the defects are hereditary, that is, fixed at the time of fertilization by reason of deficiencies inherent in one or the other or in both gametes; (2) that they are caused by the detrimental effect on growth of faulty environmental conditions, such as an unhealthy uterine mucosa or a defective placental mechanism; (3) that they may be the result of disease actively established in the fetus itself, endocarditis.

In respect to the first two theories — Dr. Streeter in writing about the early stages of embryonic development states: "There has been demonstrated to exist throughout a great variety of plant and animal forms a wide variation in the degree of vitality of seeds and eggs. Among mammals that have plural births, particularly in those having large litters, it has been shown that from 20 to 30 per cent fail to develop properly, and that the blame can rest only on the germ plasm. The evidence is accumulating that also in man the vitalities of the sperm and the ovum are factors of prime importance for normal development.

"The development of defects involves the whole egg. The egg develops and lives in proportion to the vitality with which it is endowed. Good eggs produce hardy, long-lived individuals and poor eggs succumb during uterine life, infancy, or early years of maturity. In addition to this quality of the egg as a whole, account must be taken of the varying degrees of vitality

of its different parts. In the human individual it is found that some structures succumb long before others. That this disparity in vitality of the different tissues is inherent in the germplasm is evidenced by the fact that these shorter lived parts are hereditarily transmitted to us, for example, the hair follicles of the scalp. There is a range within which the disparity may be regarded as normal. When it is beyond those limits we enter the domain of the abnormal and the pathological. Accepting this viewpoint one should be prepared to meet, in the fetus, focal degenerations and arrests of development, of one sort or another, which are not to be directly attributed to infections, nor to physical or chemical disturbances but to the fact that the original vitality of those parts was sufficient to carry them but part of the distance along their course of development, at which time they either remain stationary in whatever state they have attained, or break down and disappear. Is it not possible that many of the cardiovascular defects are to be explained as well by the constitution of the germ-plasm with which they started life, i.e., the inherited vulnerability of their vascular system, as by the much-blamed mental and physical strains, alcohol, and syphilis, which in other individuals do not result in comparable damage?

"On the other hand, in spite of the importance of the inherent quality and vitality of the embryo, we must not lose sight entirely of the environment in which it is developing. More particularly in the later months of fetal life the influence of the health and state of nutrition of the mother becomes significant."

An illustration of the environmental factor is found in the anomalies which appear in the fetus following roentgen-ray treatment of pregnant mothers.

Concerning the third theory, i.e., the effects of disease, although rare, almost every conceivable form of acquired cardiovascular disease is met with at birth, or is, in other words, congenital. An excellent account of fetal endocarditis is given by Fischer. At the Infants' Hospital we have had one proved case of fetal endocarditis and one doubtful case. It may be of interest to read the following abstracts of these cases, which were prepared by Dr. Farber.

CASE 1. A. L., 17,351, A-31-153, aged 2½ months, was brought to the hospital because of blue spells after feeding. There was a definite history of influenza in the mother, two months before delivery. There was a mild, terminal bronchopneumonia and a small amount of terminal pleurisy in this case. The main findings were in the heart which weighed 70 grams (normal 23 grams). The right ventricle measured 10 mm. in thickness (12 mm. normal for adults, and 2-3 mm. normal for this age). The right auricular myocardium was also thickened, measuring 6-7 mm. (1-2 mm. normal for this age.) There were no congenital defects in the heart or in the vessels leaving the heart. The right side of the heart showed nothing else unusual. The left auricle was distinctly thicker than normal, and there was a small amount of stenosis of the mitral valve. The valve leaflets were yellowish in color and showed definite thickening of the endocardium which covered their surfaces. There were no vegetations. The endocardium lining the left auricle also showed this peculiar discoloration and thickening. The chamber of the left ventricle was distinctly enlarged and "hollowed out". The aortic valve showed distinct stenosis and fusion of the valve leaves which were slightly nodular. The whole surrounding ring of the aortic valve tissue was very irregular, firm in consistency and undoubtedly this had caused a considerable amount of obstruction to blood passing into the aorta as well as the insufficiency of valve cusps. Microscopically there was marked thickening of the tissue of the valve with hyalinization. The papillary muscles and myocardium were diffusely scarred and showed numerous areas of hyaline changes in the muscle fibres. Fibrosis was diffuse and there was a considerable amount of separation of the fibrils. Localized areas of degeneration with numerous small areas of calcium deposition were found in the papillary muscles.

Because of the evidence of old inflammation, fibrosis, degeneration, calcification, and scarring, and because of the fact that these lesions were found in the left side of the heart, a site rarely involved in a congenital cardiac disturbance, the conclusion from the gross and microscopic findings was made that we were dealing with an infectious process, a true endomyocarditis, and because of the age of the patient, we concluded that this infection must have taken place before birth. The past history of the patient was also negative for any acute disease that might cause this type of lesion. These findings link up admirably with the history of influenza in the mother, two months before delivery. The similarity to Fischer's case in the history, and gross and microscopic findings is striking.

CASE 2. Baby Boy L, aged 3 days, 15,836, A-30-9. The main finding was a mal-formed heart with an under-developed right ventricle represented by a tiny slit and a large, calcified mass obstructing the tricuspid orifice and extending from the right auricle into the right ventricle. The heart weighed 33 grams (normal 17 grams).

Microscopical examination of the entire mass revealed nothing but calcified tissue. There was no clue as to its true nature. The theories may be advanced: I. The calcified mass resulted from the degeneration of a previous tumor. II. The mass may represent the end stage of a localized area of infection. Examination of the remainder of the myocardium did not give any additional evidence in favor of the latter theory, however.

Instances of congenital derangements of the cardiovascular system are included in nearly all textbooks, with a distinction made between the heart per se and the vascular system. L. de la Ruf reports a case of aortic aneurysm in a five months' old baby and Hochsinger reports a case at birth. Hale reports a case of arteriosclerosis involving all the arteries. Kryzyskoski reports a case of periarteritis nodosa in an

infant two and a half months of age. Also, we have seen streptococcus viridans infection in two infants with congenital anomaly of the heart.

As McCullouch says:

"If a distinction can be made between the underlying causes of congenital cardiac disorders, namely the inherited vulnerability of (1) the cardiovascular system, (2) the environment in which it is developing and (3) the effects of disease, it seems an uncritical use of the term congenital cardiovascular disease to apply it to such cardiac defects as are clearly developmental and belonging to the same category as hare lip or a persistent Meckel's diverticulum. A child with pulmonary stenosis due to an endocarditis acquired late in intrauterine life might at a given moment show the same cardiovascular symptoms as another child with a stenosed pulmonary artery caused in the second month of fetal life by an abnormal division of the truncus arteriosus. Cases are different, not only is the etiology different, but associated conditions of considerable functional importance are almost certain to be different. It would make for clearer interpretations if the word disease were avoided and such a term as anomaly used to designate abnormalities due to deranged developmental processes."

Time does not permit any discussion of the anatomic and physiologic aspects of this subject presented by Dr. Ernstene, but to those of you who are interested I would mention that Dr. Wiggers and his associates have reviewed recently the relationship of the circulatory system to growth and development for the White House Conference on Child Health and Protection. In this review they bring out many new points respecting the prenatal and neonatal circulation.

RHEUMATIC FEVER*

BY T. DUCKETT JONES, M.D.†

THE term "rheumatic fever" is an unfortunate one, and for several reasons. The fact that heart disease of the rheumatic type is found in a goodly number of individuals without a history of any joint involvement, and the fact that a recurrence of rheumatic fever may occur with but little or very low-grade fever, strongly support the feeling that some other terminology would be distinctly more descriptive of the disease process. Recurrences of rheumatic fever which are just as important a phase of the disease as the original illness show widespread signs and symptoms, and yet to date there is no definite term which will be sufficiently inclusive. There has, however, been one excellent change; we no longer modify rheumatic fever by "acute", "subacute", and so forth, since any activity of the disease itself substantiates a diagnosis of rheumatic fever.

While it has been recognized for at least two hundred years that there is actually such a disease as rheumatic fever, it was not until about 1888 following the work of Cheadle that the idea became popular that while there may be predominating joint symptoms in rheumatic fever, the major attention should be directed to the heart. Since this time there have been continually added, and very gradually, some ideas concerning the very general nature of the disease, and only in the past decade has it been generally accepted by pathologists and physicians alike that the disease is a general circulatory one. While changes which occur in the heart are extremely important and these patients often die as a result of heart disease, pathological changes may and usually do occur in very diverse parts of the body. This has been well pointed out by Pappenheimer and VonGlahn as well as by Klinge, in Germany, who have written at length concerning the blood vessel changes in rheumatic fever in organs other than the heart. More recently there has been much written concerning pleural, pulmonary, and peritoneal lesions in the disease.

During the past quarter of a century there has doubtless been some modification of the original descriptions of the disease. The frequency of acute articular, or inflammatory rheumatism seems to be progressively lessening. However, the quantity of heart disease of the rheumatic type has not lessened to any appreciable extent. There seems to be little doubt but that the disease process becomes less frequent and more vague and atypical as one proceeds to milder climates.

Since Poynton and Payne in 1900 described a streptococcus which they believed to be the causative organism in rheumatic fever, there have been many and numerous etiological agents suggested. Several individuals have described specific types of streptococci. It need hardly be pointed out that the hypothesis of a specific strain of streptococcus being causative in rheumatic fever has not been generally accepted. There has been much confusion in the literature concerning blood culture findings in this disease. Clawson and Cecil, separately, with a rather elaborate technique, have reported positive streptococcal blood cultures in a high percentage of cases. Swift, Nye, and others have failed to substantiate this finding. The theory has been advanced by Swift, Zinsser, and Bezançon and

*Read at the meeting of the New England Heart Association, Cardiac Course, at Boston, January 6, 1932.

†Jones—Instructor in Medicine, Harvard Medical School. For record and address of author see "This Week's Issue," page 553.

Weil, that rheumatic fever is a sensitization disease resulting from tissue hypersensitiveness to streptococcal infection, either through the influence of foci or by repeated infection. There is much to be said in favor of this hypothesis, though it has not been accepted by all workers. Ideas are based largely upon study of the sensitization process of streptococcal products in animals. It has not been possible in animals definitely to reproduce rheumatic fever. The frequent occurrence of a preceding upper respiratory infection and rheumatic fever has long been recognized. The close association of the hemolytic streptococcus to rheumatic fever has been pointed out by Coburn in a number of interesting experiments. The association of the hemolytic streptococcus with the varied forms of upper respiratory infection which usually immediately precede rheumatic fever, is very striking. Patients with rheumatic fever transported to Porto Rico, showed a rapid clinical improvement in a short time. The hemolytic streptococci present in their throats disappeared. Upon return to New York hemolytic streptococci appeared in the throats of most of these patients prior to recurrences of rheumatic fever. A number of other suggestive experiments have been reported, linking closely this type of streptococcus with the disease.

That the incidence of onset of rheumatic fever is greatest between the ages of five and fifteen is well recognized. It has been pointed out by St. Lawrence and by White and Faulkner, that disease incidence among several members of a family is as high in rheumatic fever as in tuberculosis. What factor heredity plays in this is as yet undetermined. It is probable that upper respiratory infection is the environmental factor of greatest importance. Susceptibility may definitely prove to be a familial trait.

It has been recently pointed out by Paul and Salinger that the mechanism of the spread of rheumatic fever through a so-called rheumatic fever family is by means of upper respiratory infection. Several members of such a family may simultaneously show varied rheumatic fever manifestations. The types of upper respiratory infection are vague and varied, such as coryza, tonsillitis, pharyngitis, bronchitis, sinusitis, grippe, and so forth.

It may be well to point out that during acute illness with evident manifestations of rheumatic fever, the diagnosis and care of the individual patient is not difficult. However, that the disease is active over a long period of time in many cases, especially in children, is not generally understood. The more frequent signs and symptoms of low-grade active rheumatic infection may be tabulated as follows:

Joint pains of varying degrees
Low-grade fever
Chorea
Various forms of erythema multiforme
Subcutaneous nodules
Nosebleeds
Leucocytosis
Vomiting
Unexplained weight losses
Prolongation of auriculoventricular conduction time by electrocardiogram
Increased sedimentation rate of the blood cells

Any combination of the above findings in a patient with a previous history of rheumatic fever or heart disease of the rheumatic type indicates definitely that the individual has rheumatic fever. The determination of the presence or absence of active disease is the most important point in caring for patients with the stigmata of old rheumatic fever. Care of upper respiratory infection of various types is also much to be desired.

DIAGNOSIS AND CLINICAL SIGNS OF RHEUMATIC HEART DISEASE IN CHILDREN*

BY WILLIAM B. BREED, M.D.†

IN considering rheumatic heart disease, it is of paramount importance to draw a sharp line of distinction between the disease as observed in adults and that seen in children. A child with rheumatic mitral disease or other evidences of heart damage presents an entirely different problem than does an adult with the same cardiac physical signs. With the latter the difficulty is largely a mechanical one and consists of the balance of innate heart muscle reserve on the one hand, against cardiac damage and bodily activity on the other. In other words, after the myocardium, endocardium and pericardium have been affected, it is simply a matter of adjustment on the part of the body to a mechanical handicap, the great variables being the extent of the damage and the congenital cardiac reserve. In childhood, however, rheumatic heart disease is almost entirely a problem of infection. The more we see of this type of case and the more carefully we observe the various manifestations of the disease, the less we are concerned with the exact valvular lesions alone, or with the extent of the damage to any part of the heart. We are searching more keenly for signs of infec-

*From the Medical Clinic of the House of the Good Samaritan.

Read at a meeting of the New England Heart Association, Cardiac Course, at Boston, January 6, 1932.

†Breed—Associate Physician, Massachusetts General Hospital. For record and address of author see "This Week's Issue," page 553.

tion than for fine points in cardiologic diagnosis. For example, a well-nourished, healthy appearing child with no signs of infection, but with well-marked mitral cardiac disease should really give us less concern than a thin, poorly nourished boy who occasionally has a small amount of fever, and shows only a systolic murmur without cardiac enlargement. Provided we can keep the first patient in good condition and free from upper respiratory infection, we can at least launch him into adult life with a definite, relatively simple, mechanical problem to work out, difficult as it may be for him personally. The second case, on the contrary, offers us nothing certain to go on. He may develop further severe heart disease and even encounter death at any time during a flareup of his latent infection. We must, therefore, look to the child rather than to the heart.

Diagnosis of typical, acute rheumatic fever is not difficult. Under the names of inflammatory rheumatism, polyarticular rheumatism, as well as rheumatic fever, a good description of the disease can be found in any medical textbook. Mere mention of the cardinal signs of such a syndrome will be sufficient at this time. A case presenting migratory joint symptoms, fever, malaise, sore throat and leukocytosis (with polymorphonuclear increase) should offer no difficulty. If within a short time evidence of carditis presents itself, the diagnosis is secure. Certainly, some such acute cases appear to be self-limited, but it has been our experience at the House of the Good Samaritan that few of them are. Accordingly, all cases are to be considered potentially of the subacute or chronic type until by continued observation these forms can be excluded. The time relationship between the initial attack and demonstrable carditis is an uncertain one—mainly because we so seldom get observations on the heart recorded at the onset, or even for months afterwards. It is in private practice only that such a relationship can be determined—under circumstances where patients are followed closely by a competent man over a period of months before and after the first attack. This we do know, however, that clinical signs of cardiac damage may appear as late as six months to a year after the onset of rheumatism.

When we come to the subacute and chronic manifestations of the disease just alluded to, we at once encounter serious difficulties in diagnosis—mainly because of its protean character and obscure signs. In this country, especially in New England, it is often ushered in with no acute symptoms of any sort. The complaints may be merely fatigue, irritability, muscle pains, epistaxis, pallor, or failure to gain weight, with nothing definite to give the lead towards a rheumatic infection. Physical examination and continued observation in the hospital, however, will usually reveal some cardiac involvement, as well as many evidences of low-grade toxemia which are of importance in treatment and prognosis. I should like now to discuss in more detail some of these factors in the light of our experience at the House of the Good Samaritan.

Joints. Affection of joints is usually of more historical than physical importance. That is to say, we read in the histories about joint and muscle pains, but are rarely called upon to treat arthralgia. It is to be said, however, that some peripheral discomfort is elicited in a very large percentage of the patients, especially in those who show other stigmata. Often such discomfort is vague, and described as nagging, muscle, or "growing" pains.

Upper Respiratory Infections. Tonsillitis appears so frequently in the histories of these children that it cannot be ignored as a factor. Even if this affection cannot be called an integral part of the disease (as was once thought) it is perhaps, along with the common cold, the greatest offender in precipitating attacks of the infection under discussion.

Fever. The normal daily range of body temperature in children has not as yet been certainly determined. Careful search through the literature reveals no accurate data in regard to normal fluctuations; but it seems to be the consensus of clinical opinion that any reading (at or) above 100° F. by rectum indicates fever. Therefore, any child who persistently produces readings (at or) above this point may be safely said to have fever, thereby giving some evidence of continued activity of infection. We have found that the highest level in the day appears between 5 and 6 P. M.

Epistaxis. This symptom we consider of great significance in the determination of continuing trouble. We have yet no definite figures, but my impression is that the incidence of nosebleed is even higher with us than that given by Coburn[1], namely 48 per cent in his study of 162 cases.

Leukocytosis. We consider more than 10,000 white cells per cubic millimeter to be leukocytosis, provided the count is made on blood drawn at about 2 P. M., and that there is a relative polymorphonuclear increase. Dr. Emmett Settle, working at the House of the Good Samaritan, discovered that *all* the patients showed the highest number of white cells in their bloods between 12 noon and 3 P. M. Frequent white cell counts are made; and no patient with such a degree of leukocytosis (over 10,000) is allowed complete freedom. It is believed by some workers that increase in the sedimentation rate is a valuable criterion of continued activity. I share such a belief, and hope that more data concerning this test in rheumatic children will be accumulated. At present we have none.

Heart Block. Prolongation of the P-R interval by electrocardiogram beyond 0.20 second indicates certainly that myocardial infection is

still present and active. Periodic records are obtained monthly on all patients; oftener, on those particularly under suspicion. In passing, it may be noted that the serious arrhythmias, such as auricular fibrillation, and flutter, are not commonly seen in childhood—although they do occur.

Nodules. Rheumatic nodules, if diligently and frequently sought, are found in many of these patients from time to time; but, having a tendency to disappear and recur they are often missed. Since they are one of our surest signs of continuing trouble, great care should be exercised in searching for them. It is not sufficient to ascertain their absence on the first examination, and thereby eliminate them from the picture. A routine examination for nodules should be made frequently, and a record kept.

Failure to Gain Weight. Unexplained emaciation and failure to gain weight present a symptom complex which we are beginning to regard as one of our important criteria of persisting rheumatic infection. When such phenomena occur *in a rheumatic child* under good living conditions, it should have important bearing on the treatment of the particular case. It is true that many of our children do gain weight while under observation—but only those who are doing well. When a patient who has been improving ceases to gain weight we at once become suspicious of a lighting up of the old flame—and search more carefully for corroborative signs of trouble.

Vomiting. Recurrent nausea and vomiting appearing in the absence of cardiac insufficiency or drug therapy, are encountered with some frequency, and are in our opinion, taken with other signs, symptoms of smouldering infection.

Skin Manifestations. Recurrent attacks of erythema nodosum and erythema marginatum, urticaria, purpura, and even petechial hemorrhages in the presence of sterile blood cultures, are definite stigmata of the disease—and should be kept prominently in mind.

Chorea. Sydenham's chorea in any form, mild or severe, is still considered a rheumatic disease, and should be treated as such. In our experience, however, cardiac complications do not have so high an incidence in chorea alone as was observed by St. Lawrence. In spite of that, nevertheless, we discharge all of the chorea patients in whom we cannot demonstrate carditis, as potential cardiacs.

Anemia. Mild secondary anemia is observed in some chronic cases, but it is not considered an outstanding feature of the disease.

Various other symptoms—mostly quite acute ones—should be mentioned, though they are by no means pathognomonic—or even indigenous to this syndrome—but do often occur during the course of the infection.

They illustrate the diversity of symptomatology encountered. Let me mention a few of the outstanding ones in the order of their frequency as reported by Coburn[1] in his study of 162 cases of the disease.

I. Fifty-eight, or 36 per cent, presented unexplained attacks of pallor lasting from a few minutes to a few days (without cardiac insufficiency).
II. Fifty-four, or 35 per cent, had severe, unexplained, at times excruciating headaches during attacks.
III. Fifty-two, or 34 per cent, complained of unexplained precordial pain, with carditis not manifest.
IV. Thirty-two, or 21 per cent, had acute abdominal pain simulating appendicitis or renal colic—without liver enlargement or cardiac insufficiency.
V. Fifteen, or 9 per cent, gave a clinical picture of acute renal disturbance by the presence of blood and albumen in the urine (no congestive failure).
VI. Fourteen, or 9 per cent, showed profuse, sour, sweating.

There is one feature of this disease that cannot have too much emphasis placed upon it, to wit, the fact that congestive failure and death practically never occur in children with rheumatic heart disease, *in the absence of a definite recrudescence of the original rheumatic infection.* That being the case, the significance of acute respiratory infections of all sorts becomes at once apparent, especially in regard to prognosis and treatment. We believe that if tonsillitis and the common cold could be eliminated, the incidence of rheumatic heart disease would be cut down to an extremely low level.

Prognosis and treatment, then, which will not be discussed at this time, rest almost entirely upon the results of careful and long-continued observation of the patient. Without the knowledge gained from such a procedure in each individual case of rheumatic heart disease no physician is in a sound position to treat such an individual child—to say nothing of venturing a prognosis.

In closing, I wish particularly to direct your attention to the infectious nature of this disease called rheumatic fever and heart disease in children, and to emphasize its chronicity. As one of my colleagues has put it, "We do not know when it starts, we often cannot recognize it when it is here, we have no cure for it, and cannot tell when it is gone."

REFERENCE

1 Coburn, Alvin F.: The Factor of Infection in the Rheumatic State. Baltimore, Williams and Wilkins Company, 1931.

TREATMENT OF RHEUMATIC HEART DISEASE*

BY WILLIAM H. ROBEY, M.D.†

WHEN we have decided that we are dealing with a case of rheumatic fever with its characteristic migratory polyarthritis as seen in the adult or its more vague signs in childhood we think of the course of treatment which we hope will hurry the disease to a termination. In a fully declared case of rheumatic fever, rest in bed comes first in the treatment and in most instances the patient's discomforts make him quite willing to accept this important part of our therapy. During the febrile stage there is apt to be considerable sweating so that a bed made up with blankets, over and under the patient, adds to his comfort. A cradle is of considerable help by keeping the weight of the bedclothes away from the tender joints. We attempt to make the joints less painful by a loose cotton dressing over a flannel wrung out in hot water and then sprinkled with a drachm of oil of wintergreen. Sometimes additional comfort is secured by placing a particularly tender wrist or hand on a splint. The oil of wintergreen dressing is an old-fashioned method of treatment but it gives sufficient relief many times to make it worth while and should not be forgotten. The patient is chiefly concerned about the painful motion of his joints. Sometimes the pain is so great that any jarring of the bed causes him agony. Pillows placed in the bed so as partially to flex the inflamed joints provide the most comfortable position.

Water should be given freely and in large amounts to replace the fluid lost in sweating. The diet should be light and easily digestible and of high caloric value. The room should be well ventilated but the temperature should be maintained at a level which will be comfortable for the patient.

When we consider the drugs which we are to use we fall back as usual upon the salicylates. If properly administered sodium salicylate, especially if combined with twice as much bicarbonate of soda, is the most efficient drug treatment at our command. The addition of bicarbonate of soda has been used empirically for many years but recently the work of Charles C. Johnson by animal experimentation has shown that the use of bicarbonate of soda, together with salicylates in full therapeutic doses, in rheumatic fever is rational. The bicarbonate for a long time has been used empirically in doses twice the size of each dose of salicylate to counteract acidosis and to prevent gastric irritation.

When we give the salicylates we hope for the best, for speedy action, for the quieting of the joint inflammation, for the cessation of the process before the heart becomes involved which in the last analysis is the only fear of rheumatic fever. We seem to have no way of telling how sodium salicylate is going to act in a given case; it may be rapid, delayed or seemingly useless. Early in my practice I was called to see an elderly man who was in great pain with tender, swollen, reddened joints. When he looked at my prescriptions for sodium salicylate and oil of wintergreen he shrugged his shoulders and said that he had tried them several times without success but he was willing to try again. At my visit the next day he was to his great surprise and mine greatly improved and he made an uninterrupted recovery. Again we prescribe the salicylates and feel as did Sir Andrew Clarke that the only treatment for rheumatic fever is six weeks—six weeks and longer as in several of our cases which lead us to enucleate the tonsils during the height of the fever and about which I shall have a few words to say later. When the compounds of salicylic acid are properly administered they act as antipyretics and analgesics. A sufficient therapeutic dose (20 grains of salicylate of soda or aspirin every two hours for eight or ten doses) comes very near to the toxic dose so that the doctor and nurse should be on the lookout for the first symptoms of toxicity which are deafness, nausea, vomiting, rarely delirium and occasionally renal irritation. I have seen a few cases of delirium from the drug. The following day the drug should be reduced about three-fourths or discontinued for several hours if marked toxicity has been induced. After that the drug should be continued at the rate of 10 or 15 grains every three hours and for about ten days after the signs of infection have disappeared. Swift recommends in some cases where the oral administration causes nausea to give 120 to 180 c.c. of a 2% solution of sodium salicylate (x grains to ℥i of water two or three times a day). Intravenous injection of the drug has little advantage. Morphia or codein is seldom necessary although I have just seen a case of acute rheumatism where the patient did not tolerate the first doses of salicylate of soda and her physician felt forced to give morphia in order to quiet her pain sufficiently to permit sleep. This case, by the way, presented an order of symptoms unusual in my experience. I have seen a number of cases which developed a bronchopneumonia during the course of rheumatic fever, but in this one the rheumatic fever followed a bronchopneumonia, in three or four days the fingers, wrists, knees and ankles being involved.

Cinchophen preparations appeared a few years

*Read at the meeting of the New England Heart Association, Cardiac Course, Boston, January 6, 1932.

†Robey—Clinical Professor of Medicine, Harvard University. For record and address of author see "This Week's Issue," page 553.

ago, attracted considerable attention and were used as a substitute for the salicylates but we began to hear of unfavorable results. Rabinowitz in 1930 reported on 50 cases with 25 deaths and 20 autopsies confirming the diagnosis of toxic hepatitis and liver atrophy as the cause of death. Toxic effects may occur at the time or as late as two months after cessation of the drug.

Realizing these dangers a purer preparation known as neocinchophen (neoatophan) and accepted by the Council on Pharmacy and Chemistry has come into use. It has a similar effect to the salicylates and is sometimes better tolerated and may be given in slightly larger doses. Some clinicians use the salicylates and neocinchophen in alternating doses with good effects. Sometimes we alternate pyramidon, an effective synthetic analgesic with salicylate of soda.

As in most infections we look forward hopefully to a serum which will shorten the course of rheumatic fever and eradicate or diminish cardiac complications. Some favorable reports have been made but the most recent observations cast considerable doubt upon its value. Swift and his associates have produced a serum but it is too early to say much about it. They think that the antiarthritic and antipyretic effect of salicylates and neocinchophen or pyramidon have been more marked than that of serum. In a few cases with acute pericarditis, serum therapy combined with drugs produced a more rapid recovery than would have been anticipated with serum alone.

No one mentions rheumatic fever without speaking of the incidence of tonsillitis and of the value of tonsillectomy. Several reports have appeared on the subject but the profession is still in doubt; yet it is generally accepted that the removal of all foci of infection is a good general principle. In this talk we do not have to concern ourselves with this problem more than to say that if tonsillectomy is performed shortly after an attack of rheumatic fever it is wise to give the patient a course of treatment with salicylate of soda or some other antirheumatic drug before and after tonsillectomy. If tonsillectomy is performed as a preventive in rheumatic fever it is well to explain to the patient that there might be a return of the arthritis immediately after the operation otherwise he may be disappointed that the very procedure from which he hoped so much has merely resulted in a continuance of the fever. As to tonsillectomy during the course of rheumatic fever we have arrived at the conclusion that if the usual medical measures fail and the disease after a few days' remission repeatedly recurs that tonsillectomy may be safely performed. Such has been our experience at the Boston City Hospital where in several hundred cases we have felt justified in attempting to bring rheumatic fever to an end. The operation has not presented any more risks than an ordinary tonsillectomy and the striking results in a considerable number of cases have offset some of the failures to influence the course of the disease. Swift thinks that the figures at present are too meager to permit a final judgment. It is probably best to give the patient the benefit of the doubt*.

The treatment of rheumatic heart disease during the course of the fever is unnecessary and unsatisfactory. We should not hurry into a diagnosis of the cardiac lesion or lesions during the height of the febrile attack, nor for several weeks after the cessation of fever. The rapidity of heart action plus edema about the valve orifices may distinctly obscure real lesions or simulate lesions in an otherwise normal heart. One of the most important conditions to note is the presence, no matter how transitory, of a pericardial friction rub for it may point the way later to the recognition of a pericardial effusion. It is a question if the salicylates ever reduce a rheumatic pericardial effusion, but I have sometimes thought that they did. Fortunately for the patient the pericardial effusion rarely requires aspiration. This is quite different from the pericardial effusions occurring in pneumonia which are usually purulent. Aspiration is quite as easy as in pleural effusion although many have far too great a fear of it.

Digitalis is seldom indicated. It is difficult to estimate the effect of digitalis upon a heart during the tachycardia of any febrile state. If the myocardium seems to be in a state of failure, small doses may be given.

The management of the convalescence requires the greatest care. No patient should be allowed out of bed until two or three weeks after the cessation of fever and other signs of active infection. There are sometimes disappointments for after three, five or seven days of normal temperature there is a return of active signs. Even after the patient has been allowed to sit up in a chair for three or four hours, there may be a return of signs or the pulse may begin to rise. If this occurs the patient must be returned to bed. If all goes well after sitting up for a few hours a day, then a few steps may be taken and the distance increased daily.

During this time the heart should be studied. A pericardial rub should direct our attention to the heart over several months to see what damage, if any, occurred. The meaning of a murmur which perhaps appeared during the rheumatic fever must be determined.

*Apropos of this is the case of a young man of 21, the brother of a surgeon who was recently seen in one of our private hospitals. He had a polyarthritis with the usual hot, tender swollen joints and fever following a tonsillitis. After a thorough course of salicylates lasting some three weeks during which a cessation of symptoms alternated with recurrences of acute joints, all foci were eliminated except the tonsils and two or three of our best laryngologists discussed the problem of tonsillectomy but expressed the fear of inducing a fatal septicemia. At last one of them consented to perform the operation, which was successfully done, with the result that within four or five days, the joint inflammation entirely ceased and over a year later the patient's brother told me that he had been perfectly well since. Fortunately, so far as could be discovered, there was no cardiac involvement, yet while the polyarthritis was going on, the attending physicians sat anxiously and helplessly by, hoping that the heart would escape.

SOURCES OF ERROR IN BLOOD PRESSURE READINGS*

BY HAROLD W. DANA, M.D.†

KOROTKOFF[1] in 1905 originally advocated the estimation of systolic pressure by auscultation, taking as a standard the point at which a sharp sound was first heard. This criterion is still being taught to some students and is still accepted by some life insurance examiners. In the great majority of examinations, this criterion is well enough, but its acceptance overlooks the possible absence of the first or second phases of blood pressure, the so-called auscultatory gap, first described by Cook and Taussig[2] in 1916. Since then articles dealing with this difficulty in measuring systolic blood pressure have appeared fairly frequently. Mudd and White[3] found an auscultatory gap present only in patients with hypertension or aortic stenosis.

It should not, however, be considered that an auscultatory gap can occur only in these conditions, for it is to be found sometimes even in hypotension. Hence I should like to stress the importance of making the blood pressure determination by palpation as well as by auscultation in every case, as is recommended by Osborne[4]. While ordinarily the auscultatory reading is from 5-15 mm. higher than the palpatory record, the latter is frequently the higher of the two, and of course is then to be accepted as the more accurate. In some patients, but not in all, palpation of the artery at the elbow is more satisfactory than at the wrist, and frequently palpation with firm pressure of the flat of all four fingers at the elbow gives the best information as to systolic pressure.

While it does not seem worth while going into the theory of whether the first weak sounds reaching the ear do or do not represent systolic pressure, if one is to rely entirely upon the auscultatory method one will fall into fewer pitfalls by taking the first return of sound, however weak, as a measure of systolic pressure, than by waiting for a sharp sound, since this point may be as much as 50 mm. below the true systolic pressure.

With regard to the criterion for the determination of diastolic pressure, there is still much difference of opinion both among physiologists and clinicians. In the article previously referred to, Osborne in 1930 advised taking the point at which all sound disappears in the stethoscope as indicating diastolic pressure. On the other hand, the majority of writers on this subject favor taking the diastolic reading at the point when the loud sounds give place to muffled sounds,—that is, at the beginning of the fourth phase.

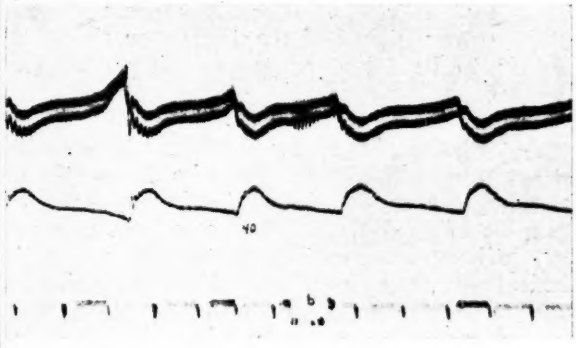
*Demonstrated by means of charts, before the Massachusetts Medical Society at the Clinic held at the Boston City Hospital, on June 8, 1932.

†Dana—Visiting Physician, Boston City Hospital. For record and address of author see "This Week's Issue," page 553.

To recapitulate the five phases briefly, quoting parts of Wiggers'[5] description: "*First Phase.* The sudden appearance of a clear sound lasting for a fall of approximately 14 mm. of mercury. *Second Phase.* The acquisition of a murmurish character, lasting while the pressure falls approximately 20 mm. *Third Phase.* The replacement of the murmur during the next 25 mm. of pressure fall. *Fourth Phase.* The muffling of the sounds, lasting while the pressure falls 5-6 mm. more. *Fifth Phase.* The disappearance of all sound."

Erlanger[6] explains the auscultatory phases of blood pressure on the basis of "hydraulic ram" effect, using an artificial circulation system with rubber tubes receiving an intermittent flow of water under a constant head of pressure, with which he reproduces all of these phases. With Erlanger's demonstration schema, however, the fourth phase is short, and there is a long fifth phase without sound.

One of the arguments in favor of accepting the cessation of all sound as a criterion of diastolic pressure, is that in certain conditions in man, such as aortic regurgitation, arteriosclerosis, etc., there is no Lower Phase of Silence, the sounds continuing in the cubital space even when no pressure cuff is compressing the brachial artery. Anyone who will take the trouble, can verify the observation of Erlanger[7] that in the dog, sounds can be heard constantly over the brachial and femoral arteries, although Erlanger considers the blood pressure in a dog as somewhere in the neighborhood of 180/120. In



(Erlanger.) Part of a mirror lever record from 3 points on a dog's artery under air compression in a glass chamber. The record of the upper 2 levers is inverted. Note the marked decrease in amplitude after beat number 39, and the disappearance of oscillation after beat numbered 40. The artery passed through a stethoscope immediately distal to the pressure chamber, and an observer signalled the falling off in the intensity of the sounds, interpolated in the time record at the point marked "b", indicating the beginning of the fourth phase, occurring at beat number 40. (See reference no. 7.)

the same article, Erlanger reproduces several sets of simultaneous "tracings" from a dog's artery passing through a pressure chamber. The "levers" recording the "tracings" were beams of light, reflected from mirrors touching the pul-

sating artery at several points, and the vessel just distal to the pressure chamber passed through a phonendoscope, the listener marking the beginning of the first and fourth phases on the record. The important part of these simultaneous records is that there is a self-evident change in the shape of the arterial impulse at these two points, and at almost simultaneous points there are signals marking the change in sounds heard through the phonendoscope indicating the onset of the first and fourth phases. In other words, according to Erlanger's records, systolic and diastolic pressures are noted almost simultaneously by changes in the sounds practically synchronous with changes in the shape of the pulse, and these changes come at the beginning of the first and the beginning of the fourth phases of pressure.

Recently I have been trying to record diastolic pressures by five different methods in each subject. The methods were auscultation, palpation, oscillation, and two others to be described, which may be called "palpable shock" felt over the armband and "auscultatory tone" heard over the armband. Of the first three methods, auscultation was taken at the beginning of the fourth phase, estimation by palpation of the greatest excursion of the pulse was usually best felt with moderate to light pressure, and was usually better made out with the pressure of four fingers over the cubital artery, while oscillation was difficult to determine accurately.

The palpable shock over the armband or armband shock is useful if present, and I have used it for some years in teaching, but since it occurs only in aortic regurgitation, hypertension or arteriosclerosis, it is not of general clinical applicability. It may occasionally be brought out in other conditions by strong compression of the air sac by the fingers. When it occurs, the lowest level at which a sharp thump or shock is felt, is to be taken as representing diastolic pressure.

The auscultatory tone over the armband, or armband sound, is usually to be heard in aortic regurgitation, but that it is to be elicited in a large percentage of patients who show no definite evidence of cardiovascular disease, was new to me until recently, and seems to offer a useful clinical correlation parallel to Erlanger's simultaneous sound change and pulse change at the beginning of the fourth phase in a dog's artery in the pressure chamber, since Erlanger compared sounds and pulsation forms inside a pressure chamber, while the clinical attempt here reported compared sounds transmitted through the pressure chamber,—in this case the air sac, —with the pulse form immediately beyond the pressure chamber, as determined by palpation or oscillation. In practice, a blood pressure stethoscope chest piece of the Bowles type, is slipped under the wrappings of the armband so that the bell lies directly outside of the air sac, close to the lower edge of the cuff, above the point where the brachial artery passes under the armband.

In some cases sounds may be heard during part of the second phase, and through all of the third and fourth phases. The lowest point of sharp sound is to be taken as representing diastolic pressure. In patients who have no cardiovascular disease, the sounds may be heard as faint beats lasting only through 6-8 mm. of pressure fall. They are never audible down to zero. To me this is further evidence in favor of taking the beginning of the fourth phase, as a measure of diastolic blood pressure.

Sometimes this diastolic tone is better heard with the blood pressure bell applied outside of the armband by means of its adjustable strap. Again, in some cases, with the bell in either position, pressure of the hand over the back of the stethoscope will bring out sounds previously inaudible, although this last manoeuvre is to be avoided if possible, since adventitious sounds may be thus produced.

In general, the measurement of diastolic pressure by these four or five methods, often agreed surprisingly closely and rarely showed variations greater than might have been due to momentary physiologic variation.

SUMMARY

1. The desirability of measuring systolic blood pressure by means of palpation as well as auscultation is emphasized.

2. Evidence is presented in favor of accepting the beginning of the fourth phase as indicating diastolic pressure.

3. A comparative study of diastolic pressure measurements in each subject by five different methods is discussed.

4. A method of auscultation over the blood pressure cuff, as a criterion of diastolic pressure is described.

REFERENCES

1 Korotkoff: Trans. Imp. Mil. Med. Acad., St. Petersburg, 1905, XI, 365.
2 Cook, Jerome E. and Taussig, Albert E.: Auscultatory blood pressure determination. J. A. M. A. 68:1088 (Apr. 14) 1917.
3 Mudd, Seeley G. and White, Paul D.: The auscultatory gap in sphygmomanometry. Arch. Int. Med. 41:249 (Feb.) 1928.
4 Osborne, Oliver T.: Medical examinations for life insurance. M. J. & Record. 132:524 (Dec. 3) 1930.
5 Wiggers, Carl J.: Circulation in Health and Disease. Philadelphia, Lea & Febiger, 2nd ed., p. 351, 1923.
6 Erlanger, Joseph: The mechanism of the compression sounds of Korotkoff. Am. J. Physiol. 40:82 (Mar. 1) 1916.
7 Erlanger, Joseph: The movements in the artery under compression during blood pressure determinations. Am. J. Physiol. 55:84 (Feb. 1) 1921.

HYPOSPADIAS AND EPISPADIAS: A PHILOLOGICAL NOTE

BY J. L. BREMER, M.D.*

THE word "hypospadias" designates, as is well known, the malformation of the penis in which there is "a congenital deficiency in the lower wall of the urethra which may terminate at the perineo-scrotal junction or at any point anterior to it". (Piersol.) "Epispadias" is described as "an absence of the upper wall of the urethra," much rarer than hypospadias. In the condition of hypospadias the penis may be curved toward the perineum, as though drawn down by the relative shortening of its under surface. Both conditions, because of the anatomical position of the urethral meatus, may cause impotency.

Embryologically these malformations may be explained by the abnormal growth or abnormal canalization of the sagittally disposed epithelial plate found in the genital tubercle of the human embryo of four or five weeks. This plate, composed of two closely applied and fused laminae, represents the earlier cloacal membrane or union of the ectoderm of the skin and the entoderm of the hind-gut, where the latter approaches the surface, similar to the oral membrane which for some time closes the entrance from mouth to pharynx. The cloacal membrane is divided, by the growth of the perineum, into the anal membrane caudally and the urogenital membrane cranially, and by the development of the genital tubercle in this region the latter is drawn out, as it were, into a sagittally disposed double plate. Normally this plate reaches the surface of the tubercle only on the caudal half of its surface, along a line from summit to base; and normally, as the tubercle grows to become the penis, the two laminae separate for varying distances along this median line, so that all male embryos pass through a stage of hypospadias. With further growth of the penis and secondary closure of the lips of the slit-like opening from the proximal end (marked by the raphé of the penis) the urethra is extended to its normal opening in the glans. A simple arrest of development, therefore, can cause hypospadias.

Epispadias, on the other hand, must result from a primary extension of the epithelial plate on to the cranial wall of the genital tubercle and a secondary arrest of development in this abnormal condition. Yet the presence of the plate on the cranial wall is in itself merely an earlier developmental arrest. The cloacal membrane originally extends from the tail-bud to the future umbilical cord, and may abnormally persist and ultimately open all along this mid-ventral line. The result is seen in cases of exstrophy of the bladder, with which epispadias is sometimes related, or with the more complete exstrophy associated with cleft penis, absence of symphysis pubis, and even absence of perineum. Epispadias is more rarely encountered than hypospadias, because it entails a partial arrest of development at an earlier age of the embryo.

In both of these conditions the external opening may vary from the normally rounded appearance of the urethral meatus (though in an abnormal position) to a long slit-like cleft, as though the two sides of the raphé had been torn apart. The edges of the slit may be irregular, still further increasing the resemblance to a tear or rent. This appearance, and a confusion in the varying meanings of a Greek word-root, have led to surprising inconsistencies in the derivations of the two words as given in the standard medical dictionaries. Perhaps, since the meaning of the terms is correctly stated, the fact that their derivation from the Greek is faulty is a matter of small interest to many, especially of the coming generation so few of whom "have had Greek". For the sake of consistency, however, and for the small number who enjoy the sensation of knowing to the fullest extent the meaning of the words they use (even though they must suffer therefor the epithet "purist") this attempt is made to clarify the situation.

The present confusion can best be shown by comparing four of the modern standard medical dictionaries. Gould gives "hypospadias" as from *hypo* and *span* (σπᾶν), to draw; "epispadias" from *epi* and *spaein* (σπάειν), to pierce. Surely the two terms are derived from the same word! Lippincott derives both from *spazein* (σπάζειν), to pierce. Stedman gives G. *hypospao*, I draw away from under, and G. *epi*, upon *spadon*, a rent. Dorland gives *hypo* and *span*, to draw; *epi* and *spadon*, a rent. It is an interesting commentary on the decline of classical learning that in the latest editions of the two latter dictionaries they have ceased printing the Greek words in their original letters. Of the general dictionaries Webster gives *hypo* + σπᾶν, to draw, tear; epi + Gr. σπᾶν, to draw; Murray, ὑπό + σπάειν, to draw, "epispadias" being omitted.

The prefixes *epi* and *hypo* have a variety of meanings in the original Greek, but usually with some suggestion of antithesis, and gradually have become standardized in medical usage to mean respectively "upon" or "above" and "under" or "below". Their use is obvious in the present case.

The Greek word-root *spa*, in various forms and derivatives, appears most commonly in the list above as the second part of the two words. *Span* and *spaein* are two forms of the infinitive

*Bremer—Hersey Professor of Anatomy, Harvard Medical School. For record and address of author see "This Week's Issue," page 553.

of the verb, and *spadon* is a noun derivative. *Spazein* as given by Lippincott is not found in the standard classical Greek dictionaries, but in the Greek-English Lexicon by Kontopoulos, published in 1900 at Athens, expressly as a guide to the modern Greek language, the word is given as "σπάξω, v. n. vul. to burst; break". It is of course very improbable, to say the least, that a compound used by Galen should be derived, with a change of meanings, from a word originating in postclassical times.

In the standard Greek dictionary of Liddell and Scott the meaning of the verb *spao* (σπάω) is given as to draw (as of a sword), to pluck out violently, to tear or rend, to snatch away; also to draw in, suck; to derive (one's name from). In medicine it came to mean to cause a convulsion, probably in reference to the violent tightening or drawing of the muscles in this condition. The word "spasm" is obviously a direct derivative with the same meaning. On the other hand the old medical term "epispastic," designating "causing a blister; a blistering agent, a vesicatory," refers more broadly to drawing in general, in this case of lymph. There is no hint that the word ever implied piercing.

From a word-root with such a wide range of meanings it is natural that the derivative words should come to connote quite different ideas. The nouns *spasis, spasma,* and *spasmos* designate respectively the act of pulling, a rag or piece torn off, and tension; but all three of them have the other meaning of a convulsion or spasm. *Spadix* is a bough or branch torn off, especially a palm branch or bay to welcome heroes. *Spadonizein* is a verb used of animals rending their prey, and the noun from which it is immediately derived is *spadon* (σπᾰδών), a tear, rent or rupture. It is easy to understand how the idea of rupture or tear should have fitted in with the occasionally wide open and ragged gap in the wall of the penis and have suggested to the lexicographers the variety of derivations given for our two medical terms; but with all due respect to the learned gentlemen, I do not think that these derivations are entirely correct.

There is another word *spadon* (σπάδων), the same as the last mentioned, when written in English letters, but differing actually in accent, as shown in the original Greek. One is accented on the second syllable, the other on the first. The confusion may have been increased by ignorance or non-observance of this distinction, as a note in Liddell and Scott's Greek Dictionary indicates that they were sometimes pronounced alike. This second *spadon,* also derived from the same root, is given but a single meaning, that of *a eunuch.*

The distinguishing characteristic of the eunuch is the loss of the testes. These viscera have been removed, and although it may seem strange to describe the surgical operation as tearing or rending away, much less as drawing, it seems certain that this meaning of the latter term has come down to us in English. The poulterer "draws" his fowls when he removes (in this case) all the viscera. The surgeon draws off the contents of a cystocele, taking away the contents of an enclosed cavity, as one also draws off wine from a cask. The question as to whether one has to deal with fluid or with solid viscera does not seem to affect the use of the word, and the implication of traction is lost. The specialized use of the word to mean the removal of the gonads only is indicated by the second Greek word *spadon* — a drawn individual —, by the Breton word "spaza" and the Welsh "dispaddu," given by Webster as meaning "to geld," and by our present use of the same word-root in the "spaying" of a bitch.

In the Middle Ages a favorite sentence for felons was that they should be "drawn and quartered". Webster's International Dictionary comments on this as follows: "As a punishment, to *draw* has designated not only (1) to eviscerate, but (2) to drag a criminal, as at the tail of a horse or cart, and (3) to tear or rend him apart, as by having each limb attached to a separate horse and the body quartered by driving the horses different ways. To be 'hanged and drawn' probably designated evisceration preceded by hanging; to be 'drawn and hanged' probably designated (2) (above) followed by hanging. 'Drawn and quartered' was used of (3) and also of drawing (1) followed by quartering."

The insertion of the word "probably" in this definition emboldens me to suggest as a possibility that the restricted use of the word "drawn" was well recognized in these times. Since total evisceration would cause perforce the immediate death of the unfortunate offender, the subsequent hanging of the body seems to me superfluous, except as an insult of which the culprit would be unaware at the time. Dragging at the cart-tail, sometimes with the addition of frequent applications of the lash, was of course a well-known punishment; but it was usually definitely described in the sentence, and was used for such minor offences that it seems hardly worthy to become part of a capital penalty. More violent dragging, as behind a horse, might result in unconsciousness or death, and thus is open to the same criticism as evisceration. Having regard to the known cruelty of those times, I am inclined to the opinion that the phrase "drawn and quartered" refers to the restricted use of the term, and implies castration. The felon was to endure the supreme mental indignity and, while still thoroughly alert, to suffer violent death.*

*D. B. Wyndham Lewis, the English historian of the times of Louis XI of France, writes: "under Elizabeth and her immediate successors the punishment for saying Mass was to be hanged until half-strangled, to be cut down alive, castrated, and ripped up, after which the heart was torn out and burned and the body quartered."

The Greek word *spadon* (σπάδων), a eunuch, was taken over directly with the same meaning into Latin as *spado*. Here, however, we find a subtle difference in connotation. The Latin *spado* is contrasted with another Latin word, *castratus*. The latter denotes strictly the loss of the testes; the former is given the more general significance of impotency, one of the results of castration. Thus in Harper's Latin Dictionary the definition of *spado* is given as "impotent, one who has no generative power (whether by nature or by castration: hence more general than 'castratus')".

In Greek, since there was only one word to express the two shades of meaning, and as this special meaning is only from a figurative use of the original word-root, it is no wonder that incongruities arose. Who would suspect that *spadix* could mean both the palm fronds for heroes and also a musical instrument, a lyre with high pitch reminiscent of the eunuch's voice, condemned by Quintilian as effeminate? *Spadonisma* means "a relaxed condition, or flaccidity," in general, with no special reference to its cause, and the verb *spadonizein* (literally to make like a eunuch) has acquired the secondary milder meaning, "to be feeble".

With the knowledge that the Latin word means an impotent, from whatever cause, and the probability that the Greek word also had the same meaning, if only secondarily, we can suggest another derivation for the two terms "hypospadias" and "epispadias". The suffix "ias" indicates the state of being, and "spadias" would mean the state of being impotent. No such word was apparently coined by the Greeks, certainly it is not to be found in the standard dictionaries; but it is linguistically possible that "hypospadias" was used to designate the condition of impotency caused by the opening of the urethra on the under side of the penis. The use of the word "epispadias" would follow naturally, when the rarer condition became recognized.

Galen recognized the possibility of impotency in this condition, as is shown in the following passage: "Those who are called hypospadiacs . . . cannot procreate, not at all because they possess no fertile sperm, but because, from the distorted condition of the penis, they cannot direct the stream properly." [liber XV, cap. III] The completion of this passage, however, indicates that impotency was not necessarily connected in his mind in the use of the term, for he says, "Those who are called hypospadiacs, because, on account of the band to the end of the penis, the meatus is distorted, cannot procreate," etc., and continues, "which fact [that they possess fertile sperm, but misdirected] is proved by its own cure, for if the band is cut away they can generate."

It is clear that here Galen refers to the downward curvature of the penis in many cases of hypospadias. It is possible that the word signifies merely "the condition of being drawn down", referring to the most obvious characteristic of the penis in these cases. The Greek word *spadon* (with the accent on the second syllable) means "a spasm or cramp" as well as "a rent or tear", and spasm, as we have seen, carries with it the idea of a drawing or pulling of the muscles.

In a kind of glossary of terms later in his works Galen definitely returns to this meaning; "Hypospadias is that malady in which the glans penis is retracted;" but he continues "or that malady in which the glans penis remains normal [lit: is left as it is] and the meatus of the urethra is spread out underneath." He obviously recognizes two types of the deformity, but the retraction of the glans is first and probably the most prominent in his mind.

We are offered then, three possible meanings for the word hypospadias, all fitting conditions known to Galen when he first used the term. It may mean "drawn under", "ruptured or torn on the under side," or "impotent because of the opening on the under side." The first two are derived from different meanings of the word *spadon* (σπᾰδών), either 'a cramp or drawing' or 'a rent or rupture;' the last from the other *spadon* (σπάδων), meaning "eunuch or impotent". Probably the simplest, most obvious condition actually gave the name to this deformity, but the fitness of the other interpretations can hardly have escaped the "laughter-loving Greeks."

A full definition of the word hypospadias might then be given thus: [from Gr. hypo, under or beneath, and *spadon* (σπᾰδών), a spasm or cramp] a malformation of the penis in which the glans is distorted as though violently drawn down underneath; or [from *spadon,* a rent or rupture] the condition in which the opening of the urethra is on the under side of the penis; or [from *spadon* (σπάδων), impotent] the condition of being impotent because of the position of the meatus on the under side of the penis. "Epispadias" should, of course, be given parallel derivations and meanings.

THE ETIOLOGY AND TREATMENT OF CHRONIC ARTHRITIS*

BY REGINALD BURBANK, M.D.†

I HOPE you do not expect me to talk this evening about any cure for arthritis, as an arthritic joint frequently shows permanent change both by x-ray and by physical examination, and while the patient may be symptom-free and can consider himself cured, permanent changes may be present in the joint which render the word "cured" incompatible with the findings.

I would like to go into some detail as to the cause of arthritis. We feel that there may be a number of contributory causes, such as a peculiar individual susceptibility to infection, glandular dyscrasia, calcium or phosphorous imbalance, mental or physical trauma and so forth, but we wish to emphasize the fact that we do not believe chronic infective arthritis of the so-called non-specific type exists without streptococcal infection. We are, of course, not referring to tubercular, gonorrheal, syphilitic, or traumatic joints. Other organisms than the streptococcus may play a part in the production of the disease, but so far only the streptococcus has been proved to be a definite causative factor.

In the early days of bacteriology, Mantle isolated cocci from rheumatic joints, and was able to produce experimentally joint lesions in rabbits. These were probably pus joints, as the rabbit is particularly susceptible to pyogenic joint infection. The streptococci these days were not differentiated, and consequently work of the early experimenters gives very little definite information. From 1900 on, many men used vaccine and it was hailed as a panacea and used so promiscuously and with so little knowledge of its effect that it probably did more harm than good, and consequently its use came into disrepute.

Doctor T. W. Hastings of Cornell was one of the first to do any really scientific work on the study of the blood serum in arthritics, and using the old inactive serum Wassermann technic, he was able to obtain positive fixation with certain streptococci isolated from arthritic patients. Single strain fixations were used, and the work was in consequence rather unsatisfactory, for certain cases gave positive fixation, whereas others, apparently similarly afflicted, would give clear negative fixations. In following up this work of Hastings, we felt that it was necessary to employ multiple strains instead of one single streptococcal strain, and for this purpose we isolated streptococci from various foci of infection, using only definitely proved arthritic strains as antigens. If a patient showed an infected tooth and the arthritic symptoms cleared up spontaneously on extraction of the tooth, we considered this presumptive evidence that the streptococcus obtained from this removed focus of infection was the causative factor in the production of the arthritis. A large series of such antigens was collected from various foci of infection, sinuses, tonsils, teeth, prostates, and so forth, and these strains were used for the complement fixation tests.

It was necessary to use active serum rather than inactivated as we found that heat inactivation destroyed the thermolabile complement, and consequently rendered many fixations negative which would have been positive with the active serum complement fixation.

In doing this work, we came to the conclusion that strains of streptococci might be biologically different even though morphologically the same and having identical sugar fermenting reactions. We found the hemolytic streptococcus the preponderant fixer in periarticular arthritis, the viridans streptococcus the one which gave fixation in osteoarthritis. Most cases seen, however, were of rather long standing and were of mixed type, and consequently gave fixation with both hemolytic and viridans streptococci.

These complement fixation tests varied from time to time according to the condition of the patient as influenced by superimposed infection. For example, an osteoarthritic giving a pure viridans fixation might have a grippy cold or tonsillitis, and with or without developing periarticular joints, a repeat fixation might be positive with both viridans and hemolytic streptococci, the latter due to the superimposed infection.

The presence of antibodies in the blood stream of an arthritic presupposes the entrance of corresponding streptococcic antigens into the patient's blood stream, and our next step was to see if positive blood cultures could be obtained in afebrile arthritics. For a number of years no positive blood culture findings were obtainable. In 1925, however, we were able to obtain positive blood culture findings in these afebrile arthritics by inactivating the patient's complement, thus allowing the organisms to grow. Eighty per cent of these positive cultures came under two groupings according to sugar classification. The hemolytic type were infrequens, the viridans, faecalis. Having obtained the organisms from the blood stream, we attempted to isolate them from arthritic joints but only occasional cultures were obtained from the joint fluid, and when a culture was positive we usually found some tissue present and gradually came to feel that the tissue was the lodging place for these streptococci rather than the joint fluid itself. The cultures obtained from these tissue

*Read at the Boston City Hospital Clinic, December 18, 1931.

†Burbank—Chief of Arthritis Clinic, Cornell University Medical College 1916-1918. For record and address of author see "This Week's Issue," page 553.

scrapings corresponded very closely to those obtained from blood cultures.

Our next step was to inject these blood culture organisms into animals and see if a low-grade indolent arthritis could be produced. Previous experimental work in animal inoculations had produced pus joints rather than low-grade arthritic joints. Using blood culture strains, the animals at first showed no change except a slight loss of weight during the initial septicemic stage. This lasted a few days, then the rabbit apparently came back to normal, but at intervals varying from a week to several months, progressive joint involvement became evident. At first, the rabbit would protect the joint, but no visible signs of arthritis were present except for a slight swelling demonstrated by x-ray. This was the periarticular stage of involvement. Later, a degenerative and regenerative stage was apparent, easily demonstrated by x-ray. Round cell infiltration was present. Pus cells did not occur. Occasionally, these experimentally produced lesions would clear up spontaneously, but in most cases the joint involvement became progressively severe. At no time were pus joints present, and throughout the entire series of experiments, the rabbits lived normal lives and finally were killed for joint study.

The next step in proving the culpability of the streptococcus was to show the actual dissemination of the organisms in the joint tissues, and this phase of the work we are going to show you this evening. The slides will demonstrate the streptococci in chain formation in the joint tissues themselves, to our mind definitely proving that the streptococcus is the primary infective cause of arthritis and that the actual distribution of the organisms is responsible for most joint manifestations rather than allergy or toxicity.

The majority of cases of arthritis which we see start in with some history of infection and we feel that a basic infection is necessary to bring on the disease, coupled in all probability with some predisposition on the part of the patient to streptococcal infection. The majority of cases seen by us are of long standing and consequently have had their major foci of infection removed and yet the disease persists.

We make a routine examination of the patient, culturing any apparent focus. We feel that many a beefy, congested throat may be just as much a source of systemic absorption as an infected tonsil. We also feel that chronic gingivitis and pyorrhea may give systemic absorption as well as apical abscesses.

It has been our custom to make routine stool examinations on all cases that come to us and it is surprising to find that in more than ninety per cent of these cases streptococci of either faecalis or infrequens types are present. The stool cultures corresponded so closely with our blood culture findings that we felt adequate care of the intestinal tract was essential in the proper treatment of these arthritics. Our feeling is that the primary focus of infection may have left a secondary implant of infective organisms in the intestinal tract, and even though the primary focus be removed, the secondary implant continues to give absorption and consequently acts as a secondary focus for the arthritis. Both the infrequens and faecalis are facultative anaerobes, and consequently can carry on their life cycle in the intestinal tract without the presence of any free oxygen.

Each new case that comes to the office has a routine complete bacteriology of all foci, the upper nares, the pharynx, the tonsils, the teeth, any bronchial expectoration, prostatic or cervical cultures if indicated, and routine stool and urine cultures. Six to ten c.c. of blood is taken for the complement fixation work, for a routine blood culture, and for blood chemistry if indicated. A general physical examination is also done at the first visit. Palliative measures are instituted if necessary and the patient told to return in a week's time after the bacteriological and serological examinations have been completed. If the patient's blood shows an adequate complementary titre, a vaccine is made up and the patient put on such dosage as we feel he is able to tolerate. If the disease is acute, or the patient's resistance low, minute doses are given, sometimes not more than fifty bacteria per injection. We do not feel that reactions are either necessary or advisable in the treatment of these cases. If no complement is present, vaccine is contraindicated, and it is necessary to build up the patient's general resistance. Symptomatic improvement may be obtained in these deficient complement cases by giving very small doses of specific toxin rather than antigen.

I am anxious to impress my belief that arthritis is not a disease of the joints, but is a systemic disease and that it is necessary to treat it as you would treat any other chronic infection. If the patient is run down, hematinics and general tonics should be given. Such foods as the patient does not digest should be eliminated from the diet, but diet should not be restricted to the point of depleting the patient. Sweets and highly seasoned foods are as a rule eliminated because the former furnish too good a medium for the growth of the streptococcus and the latter, because of their irritant action, increase absorption from the intestinal mucosa. The patient's bowels should be kept well open with at least one full free movement per day.

We urge the patient if possible to keep up and about as it is an extremely difficult thing to get a chronic arthritic out of bed again once he has been bedridden. We make it a cardinal rule to advise exercise, but never to take an amount that will make the joints hurt worse after the exercise is finished. Sunshine, fresh air, deep breathing, orthopedic care for correc-

tion of deformities and any other indicated measures are advised. We do not feel, however, that exercise, posture or the ordinary orthopedic procedures alone can clear up an active arthritic involvement.

As regards dosage of vaccine, we cannot stress too strongly the fact that it is essential to keep below reaction point. Severe reactions only increase the patient's toxic load, and are distinctly unfavorable, particularly in severely afflicted cases. If a patient has a reaction from an injection, we cut the dose down to one-tenth. If this lowered dose still gives a reaction, we cut down to one-hundredth of the original injection, and in many cases we find that the best results are obtained on doses varying from fifty to five thousand organisms per injection.

There are many theories in addition to the bacterial as to the causation of arthritis, but until some one is able experimentally to produce arthritic lesions with regularity and uniformity with some other technic than that of streptococcal injections, we will not alter our viewpoint—that selective strains of streptococci are responsible for that systemic disease known as multiple infective arthritis.

Dr. Hadjopoulos then showed a series of lantern slides demonstrating the pathology of experimentally produced joint lesions and the actual distribution of the infecting streptococci in all the tissues that are affected in the disease.

Discussion

Dr. George P. Sanborn: From available evidence, clinical, laboratory, and from animal experiment, it seems reasonable to regard rheumatoid arthritis as an infectious disease; focal infection as the source; streptococci emanating from the foci, the inciting agents; the blood stream the carrier of streptococci to the joint tissue. Focal infection, streptococcemia, and susceptibility of joints to streptococcus invasion, are the requirements for development of infectious arthritis. To be somewhat dogmatic, in the absence of any one of the three, rheumatoid arthritis cannot develop.

Focal infection is common, but streptococcemia and arthritis develop only in comparatively few. Excluding the exceptional cases in which situation of the focus or virulence of the streptococcus accounts for invasion, in general it may be said, that the development of streptococcemia and arthritis is determined by susceptibility to streptococcus infection derived from predisposing (hereditary, constitutional, occupational) and precipitating causes. Predisposition probably amounts to having a vulnerable immunizing mechanism, that breaks down under stress of precipitating causes, such as exposure to cold and wet, exhaustion, physical and mental shock, and so on. Study of predisposing and precipitating causes in the given case is of great importance as a basis for successful treatment.

Therapeutic procedure in infectious arthritis depends on the condition of the patient, as revealed by painstaking physical examination, and on facts revealed by careful history. Rest in bed may go far in eliminating from further activity, precipitating causes; to some degree may modify the effect of predisposing causes; but quite as important, it should lessen the amount of auto-inoculation from infected joints, which active use of the joints may induce.

Search for inciting causes, that is, focal infection, and removal or drainage of foci, as soon as the patient's condition permits, is of paramount importance, and in the early months of the disease may result in complete recovery; but if the disease is of years' duration, such a favorable result is not anticipated.

Cultures are made from all foci removed or drained, and as many strains of streptococci as possible isolated. Cultures should also be taken from the blood, joint fluid—if possible, enlarged lymphnodes—if any, urine, feces, prostate, and from postnasal space. All strains of streptococci that are agglutinated by the patient's serum, or fix its complement, or yield a positive cutaneous reaction when injected into the patient's skin in small dosage, should be preserved, or made into vaccine for use later, if necessary.

An autogenous vaccine including in it all the strains of non-hemolytic streptococci that have been derived from foci and elsewhere, is usually satisfactory. If some strains are inert it will make little difference. In addition it is usually of advantage to use a stock streptococcus vaccine of many different strains that have been obtained from foci in other arthritic cases. Dr. Burbank's method of selecting strains for a vaccine is an excellent one in his hands, in that it cuts down the number of strains to a minimum, and avoids the use of many that may be inert. Such a vaccine is an ideal one to use.

It is unnecessary to place any further emphasis on the idea that at present seems correct, namely, that multiple foci, and a multiplicity of strains of non-hemolytic streptococci may be active in a given patient.

In chronic infectious arthritis persisting after obvious foci have been removed or drained, attention has been directed to the intestinal tract as a possible focus. Treatment by promoting free bowel evacuation, intestinal antisepsis, and the use of vaccine from streptococci found in the feces, has appeared to be effective in some of these cases. The types of streptococci found in the feces may resemble pharyngeal strains, and suggest implantation from that situation.

Vaccine therapy should not be instituted while the patient has much fever, or is greatly prostrated; at any time before foci are removed or drained (unless none can be found); or if his

blood is known to be deficient in complement. Generally, vaccine should be withheld until it is quite certain that removal or drainage of foci is not going to be followed by favorable result. It hardly seems necessary to insist that every medical and orthopedic indication be met.

Arthritic cases are usually sensitive, and often very sensitive to a correct vaccine, and unpleasant reactions may be induced, which, if repeated, affect unfavorably the general condition of the patient, and the status of the disease. On the other hand, much smaller dosage in the same case may be followed by a favorable response, improvement in symptoms, and feeling of well-being. Dosage should be increased from this point with the idea of producing favorable response after each inoculation. A gradual gain of weight from week to week may be taken as evidence that all is well with the patient; loss of weight suggests too much vaccine, unless some other cause for it is apparent.

Finally, it is possible to regard patients with chronic infectious arthritis as subject to an acquired allergy to streptococci, and to consider that repeated inflammatory reactions of an allergic type in the joint tissue are, at least in part, responsible for the pathology, not excluding, of course, the possible effect of toxin derived from streptococci within the joint. Certainly, the type of vaccine procedure required in treatment of these cases suggests desensitization as an important issue.

DR. A. G. HOWARD: In 1923 I started a clinic for the study and treatment of arthritis following the technique of Doctor Burbank at the Massachusetts Memorial Hospitals. This was after we had observed the wonderful work accomplished by him in his New York clinic. We endeavored to follow his method accurately, and checked on our results in as many ways as possible. The results were and are very satisfactory in a large proportion of the cases, and we are enthusiastic over the end results.

We were forced to discontinue the clinic at the end of the third year because of the expense to the hospital, and therefore were unable to check some of these cases. In private practice all of the cases are rechecked by several Burbank tests at intervals of several months or a year after the patient has become symptom-free and presumably immune. The clinical results have been extremely gratifying in a large percentage of the cases. The value and specificity of the Burbank treatment seem to be further proved by the great percentage of negative Burbank examinations after the patients have become symptom-free. Even so, operative treatment should be delayed for a considerable period because in one case in which the disease appeared to have been arrested all symptoms returned after a synovectomy of the knee which was probably done too early.

DR. WILLIAM H. ROBEY: I find that I am one of the few medical men in a large group of orthopedic surgeons who have come to listen to Doctor Burbank's paper describing his methods for the relief of chronic arthritis. The disease is fundamentally medical, but because so little has been done for it by physicians, it has really become classed as a surgical disease. In other words, its active treatment becomes orthopedic because the orthopedist is finally called upon to correct painful and disfiguring deformities.

Doctor Burbank has not neglected to stress the importance of removing foci of infection. We have had considerable experience in this hospital with the removal of tonsils for various rheumatic conditions, and have up to the present time done a little over five hundred tonsillectomies during the height of rheumatic fever. We have also found a number of cases with tonsillar remains. Of one hundred and forty-three patients who had tonsillectomies at the Massachusetts General Hospital, tonsillar tissue was found to be remaining in fifty-nine. A routine physical examination of the nurses entering training in the Presbyterian and Cook County Hospitals disclosed fairly large pieces of tonsillar tissue remaining in two hundred and ninety of four hundred and three (seventy-three per cent) who had had tonsillectomy. In the Boston City Hospital of two hundred and one patients, fifteen per cent showed tonsillar remains of varying degrees. Tonsillectomy should be performed only by those who have had special training for the operation.

During early life rheumatic fever produces endocarditis in the young when the blood vessels of the valves are larger and capable of carrying the infecting virus to them. This danger steadily diminishes as the blood supply of the valves definitely lessens in later life. Chronic arthritis does not present this danger, but merely the pain and deformities which come on gradually, and because of their interference with the general health of the patient, lead to a final breakdown.

DR. BURBANK (Closing the Discussion): There were some very interesting points brought up in the discussion. Doctor Sanborn said that we were doing all that could be done. I wish we were. We are doing the most that we know how to do with the funds we have. We are not yet satisfied with the work we have done, but would like a complete check-up from all other points of view. There are a lot of cases that we cannot hit.

In our last series of 1000 cases observed for more than a period of two years, ten per cent were worse or unimproved. We do not believe it is the fault of the principle of treatment. It is our human limitation in not knowing enough about it yet.

There are other complications which enter into the treatment in certain general systemic diseases, such as diabetes, tuberculosis, cancer and to a less extent syphilis.

These diseases make treatment very difficult and an enormous amount of patience on the part of both the doctor and the patient is required.

Doctor Robey brought out the advisability of enucleation of tonsils in rheumatic fever. The younger the patient, the more apt you are to clear up the disease by enucleation of the foci. The older the patient, the less likely you are to do so. We feel that the intestinal tract and the joints are the major secondary foci and for that reason we believe that these require consideration before you decide upon operative procedure on arthritic joints. I know of many monoarticular joints operated upon with polyarticular involvement set up. We feel that any joint which has symptoms of activity should be given every possible opportunity for recovery before operation is done

You brought up the fact that the same type of organism may cause acute rheumatic fever and arthritis. The difference probably is due to the difference in the immune reaction in the patient. The younger the patient the less immunized to streptococcus infection. In youth it hits in an acute way with a tendency to return of joints to normal. Heart involvement is of course frequent in acute rheumatic fever, very much less frequent in arthritis, but we do see it in arthritis of middle age. The more acute the joint as a general rule, the younger the patient, the more quiet and indolent the joint, the older the patient. For this reason, we see more periarticular types before the age of forty, more viridans types after the age of forty. The average case that we see with incomplete tonsillectomy has definite signs of focal infection with red anterior pillars. Chronic congested beefy throat is a sign of definite infection of the nasopharynx. The normal nasopharynx does not allow much systemic absorption, an irritated one does. In treatment of Still's disease, we have had good results. Some years ago, we presented a series of twenty-seven cases at the Babies' Hospital. At puberty, Still's disease may exacerbate and even quiescent cases may recrudesce. With the exception of four cases, we had good results in all our Still's. One boy came back who was treated at Cornell in 1916. He was then sixteen months old and he was a perfectly typical Still's with glandular enlargement with febrile reaction and the multiple joint involvement. He cleared up entirely and we did not see him again until the middle of last year. He had a recrudescence but cleared up very satisfactorily again. Several other cases have given a similar history. Not infrequently on finding them starting up at puberty the average results are excellent.

Doctor Howard showed more than sixty per cent of arthritics that had become symptom free and given negative complement fixation after becoming symptom free. It is important to warn patients that any recurrent infection may bring back a positive fixation even though they may have remained negative for a long time and that symptoms may recur with return of absorption from any superimposed infection.

PILONIDAL SINUS*

A Review of 120 Cases

BY FRANK GLENN, M.D.†

PILONIDAL sinus, or coccygeal fistula, is frequently met with on all surgical services today. It is a state completely resistant to conservative treatment and by no means readily curable even by radical surgery. Accounts of it in the literature, up to the present time, are remarkably few. To a brief summary of them is added here a report of 120 cases from the surgical service of the Peter Bent Brigham Hospital.

Writing on this subject in 1923, Dr. Harvey B. Stone[1] of Baltimore gave a full review of the then existing literature and the following excerpts are taken from his paper. J. M. Warren first discussed pilonidal sinus in 1867, describing in detail two cases and referring to ten others that he had seen in practice, under the term "inflammation of the lower caudal segment". In 1880, Hodges gave the name, "pilonidal sinus" (nest of hair), and offered as its etiology the invagination of hairs in the postanal fold. In discussing "postanal dimple", "pilonidal sinus" and "postsacral dermoid", Couraud, a Frenchman (1883), considered all three to be various degrees of the same process and quoted several explanatory hypotheses. He stated that Kuhn held the sinus to be a remaining trace of spina bifida, while Lawson Tait considered it due to the evolutionary disappearance of the caudal appendage. He credited Féré with explaining its origin as a failure of the two halves of the body to unite. Soon thereafter, in 1885, Wendelstadt reported a few cases and offered the theory that, because the inferior end of the spinal cord is the last portion to lose its connection with the skin and because the sacrum closes together and pinches off the connection, the skin at this point is left attached by fibrous bands to the posterior surface of the sacrum, and forms a dimple or pocket as the surrounding parts grow out. Her-

*From the Department of Surgery, Peter Bent Brigham Hospital, Boston, Massachusetts.

†Glenn—On staff of the New York Hospital, New York City. For record and address of author see "This Week's Issue," page 553.

man and Toureaux, in 1887, pointed out that after the separation of the spinal cord from the superficial tissues a process of epithelial cords or tubules persists for a time at the point of previous connection between the neural axis and the skin, forming a vestigial remnant. This normally disappears, but when it persists, may result in the formation of a pilonidal sinus. Stone[2], in 1931, again came forward with a new theory. The preen gland or oil gland of birds, situated near the caudal end of the vertebral column, produces an oil-like substance, is lined with epithelium and frequently contains a small tuft of feathers. This corresponds to the location of the pilonidal sinus in man. The similarity of structure of the skin lining the pilonidal sinus of man with that of the preen gland of reptiles and avians suggests that these are analogous structures.

The common site of the lesion is the posterior mid-line of the body in the sacrococcygeal region. A dimpled orifice is usually seen, from which a tubular, duct-like passage leads into the underlying tissue. The passage, which usually but not always can be entered with a probe, runs upwards, and sometimes for a lesser distance analwards, to a varying depth, ending blindly at from 1 to 6 cm. from its origin, but never actually reaching the periosteum. The orifice in the mid-line is usually single but may be multiple, is smooth, rounded and varies in diameter from a small dimple to a size of 1-5 mm. From this sinus there sometimes protrude a few hairs. The tract is lined with modified stratified squamous epithelium containing hair follicles, sebaceous glands and sweat glands. It is frequently filled with the products of the glandular structures of the skin, that is, caseous material, hair and exfoliated epithelium.

The structure of the sinus explains its clinical importance. The active sebaceous and sweat glands fill the tract with irritating material; the growing hairs may be forced directly into and through the skin, or curl about to fill the cavity. Once the continuity of the cutaneous lining is broken, infection gains entrance and persists. Inflammation closes the outlet and abscess formation results, tending to recur until the involved area has been completely excised. Clinically the patient is seldom aware of the existence of this lesion until infection has taken place. This occurs, as a rule, in early adult life, some time after puberty, when the productive structures of the skin become active. Opening the pocket, save that it gives temporary relief from the infection, leaves the basic lesion intact. In some weeks or months the abscess recurs, breaking out again, perhaps, in a new place or even in several places, often at a distance from the mid-line.

In the meantime, whether the lining of the sinus has been penetrated by growing hair or by rupture due to the pressure of the material contained or by the infectious process itself, epithelium often grows out from it in bizarre-shaped pockets and tracts, for several centimeters perhaps, in one direction or another. As a rule, this epithelium is confined to a more or less indurated zone, ending in a lateral opening, but it does not always follow so evident a course. It may extend well out into the subcutaneous tissues, in one or more places, without giving any external sign of its presence, and in such a case, unless a wide excision is performed, pockets of skin are left to reproduce the lesion. Thus the disease has a tendency to recur locally, almost like a new growth.

The most common and frequent symptom noted by the patient is tenderness and swelling over the lower sacrococcygeal region. Usually a median swelling appears and the occluded sinus may open to discharge a foul smelling material containing the products of infection. Then swelling and soreness for a time subside. The degree of pain is usually negligible in the beginning but, if the process is allowed to continue untreated, may become sufficient to prevent the patient from going about his regular duties.

Pilonidal sinus is to be distinguished from fistula-in-ano by its location, directly in or very near the mid-line, and by the course of its tract or tracts which lead away from, rather than toward, the anus. Osteomyelitis of the underlying bones of this region, rare in any case, can be excluded by examination of the termination of the sinus tract with a probe. If osteomyelitis is present, roughened bare bone will be encountered and further evidence can be had by examination with the x-ray. Any recurrent abscess in the mid-line, over the coccygeal region, should be considered a pilonidal sinus until proved otherwise.

Of the 120 cases reviewed, 23 were in females and 97 in males, or 19% females to 81% males. Twenty-two of the males were of known Russian-Jewish descent. The average age for the onset of symptoms was between 22 and 23, the youngest patient being a girl of 17 years. The average duration of symptoms prior to hospitalization was from two to three years, although in one instance, that of a 64 year old man, the symptoms had been present for 41 years! Almost without exception, various methods of conservative treatment had been employed, as, for instance, hot sitz-baths, cauterization of the sinus tract with phenol, silver nitrate, nitric acid or other caustic agents, simple incision with packing of the cavity with iodoform gauze or repeated swabbing with tincture of iodine. All of these gave the patients, according to the histories, considerable relief of symptoms for periods of days and sometimes months.

Trauma in some form was almost invariably given as the inciting factor by the patients, and symptoms dated from such incidents. This was probably only attempted rationalization on their part. However, falling and striking the coccyg-

eal region, long and frequent automobile rides, horseback riding and direct violence were most frequently cited as preceding the onset of symptoms.

Contrary to a statement sometimes seen in textbooks, that congenital anomalies accompany pilonidal sinus, only two cases in the entire series of 120 (19 were examined by means of the x-ray) were found to have such—spina bifida occulta in both instances.

Various methods of treatment were employed in this series of 120 cases. In 82 cases, block excision was done and the wound closed without drainage. In 26 others, after excision, the wound was packed open and allowed to heal by granulation. Nine cases, for one reason or another, were merely incised and drained. Three patients left the hospital after refusing operation. Of the first group of 82, in which the wound was closed, only 36 healed by primary union; the remaining 46 showed evidence of infection and the wounds reopened, to heal later by second intention.

End-results are available for the first 100 cases over a period of two years or more after leaving the hospital. Of these, 40 made no reply. Of the rest, 43 were completely relieved of symptoms and 17 suffered recurrences, returning to the hospital for further treatment. As to the 40 that made no reply, it is logical to assume that many had recurrence of symptoms and sought treatment in other institutions.

The average period of hospitalization for each case in the entire series was a fraction less than 13 days. Some of the recurrent cases were in the hospital for a total period of 90 days or more, followed by a long period of attendance for treatment in the out-patient department. Prolonged hospitalization was usually the result of inadequate and too conservative treatment.

Treatment by block excision after the injection of the sinus tract with methylene blue has been found to be the most satisfactory method, care being used to cut well beyond the extension of the dye. It is desirable to have the aid of the pathologist at the time of operation to corroborate the surgeon's findings and to search for evidence of small cord-like structures; for at the very end of the sinus tract or tracts, small cords or pockets are often seen, becoming smaller as they extend into the surrounding subcutaneous tissue. These structures, lined with or composed of cells, as has already been explained, are not only the seat of recurrent infection but, if not completely excised, remain to reconstruct the lesion. If it were not for them, the cure of pilonidal sinus would be a trifling matter. The area of skin incised need not be large but undercutting is necessary to include a sufficiently large area within which the arborization of the process may be expected to lie. Usually about midway between the skin and the sacrum the sinus pattern is the largest. This area and the tissue surrounding any gross lateral sinus tracts and openings which may be present must be completely removed to insure a cure. In the performance of this operation, excision of the fatty subcutaneous tissue down to the periosteum is absolutely essential.

Closure of the wound in layers, after exact hemostasis, with suture material sufficiently strong to secure a complete obliteration of dead space, is fundamental. Black silk is well adapted to the closure of the skin. Drains should never be left in the closed wound. If there is fear of infection or if there is reason to believe that the sinus tracts have not been completely excised, then the wound should be packed widely open with gauze.

Postoperative care of the closed wound is of great importance and often determines the incidence of wound infection. It has been found very satisfactory to apply a silver foil dressing over the incision and to place over this a gauze dressing, held in place by adhesive plaster so arranged as completely to seal off the anal region and to diminish the tension placed on the sutures by allowing the strips of adhesive to extend well out over the gluteal regions. The patient is given a diet of constipating nature so that movements of the bowel may be inhibited for a period of 6-7 days. His position in bed is of significance. He should, if possible, be kept on the side, the face slightly downwards and legs straight, so that at no time will the sutures in the wound be subjected to increased tension. The gauze dressing overlying the wound may be kept moist with alcohol by adding 10-15 c.c. every four to six hours, for the first 24 hours. In this way, an attempt is made to prevent infection of the incision from external sources.

Should the wound become infected, the period of hospitalization will be decidedly shortened by removing all sutures, opening the wound widely and inserting gauze packs soaked with some such antiseptic material as dichloramine T. or Dakin's solution. Recurrent cases are more prone to postoperative infection than those operated upon for the first time, and it follows also that in such instances the margin to be excised beyond the tissue evidently involved should be far greater than usual.

SUMMARY

(1) A review of the theories of the origin of pilonidal sinus is inconclusive.

(2) The infected pilonidal sinus is of clinical importance.

(3) A review of 120 cases is given.

(4) Incomplete excision of the entire sinus tract and its arborization results in recurrent symptoms necessitating further treatment.

REFERENCES

1 Stone, Harvey B.: Pilonidal sinus. Ann. Surg. 79: 410, 1924.

2 Stone, Harvey B.: The origin of pilonidal sinus. Ann. Surg. 94: 317, 1931.

CASE RECORDS
of the
MASSACHUSETTS GENERAL HOSPITAL

ANTE-MORTEM AND POST-MORTEM RECORDS AS USED IN WEEKLY CLINICAL-PATHOLOGIC EXERCISES

EDITED BY RICHARD C. CABOT, M.D.
F. M. PAINTER, A.B., ASSISTANT EDITOR

CASE 18381

ABDOMINAL PAIN, DIARRHEA AND COMA

MEDICAL DEPARTMENT

PRESENTATION OF CASE

DR. ALFRED KRANES*: *First admission.* This is the case of a schoolboy twelve years old when he first entered the hospital.

He had previously been well until he suddenly noticed that his left foot, later the whole left leg, became very weak and collapsed under him when he attempted to stand. Shortly after that his left arm became useless. Progression occurred quite rapidly for fifteen minutes, then stopped. A doctor was called. Within the next three hours the patient vomited four or five times.

The past history was that he was born after a very prolonged labor lasting about twenty-five hours, ending with a forceps delivery. He developed perfectly normally. At eight he was run over by an automobile. He said that the wheels went transversely over his abdomen. He remained in bed eight days. X-rays were negative. There appeared to be no ill effects from the accident except that since then he had had nocturnal enuresis. Six months after that accident he had a period of jaundice which lasted three or four days.

On admission to the Emergency Ward it was found that the whole left side was paralyzed. He was conscious and could answer questions, but only after some prompting. His neck was not stiff and there was no Kernig. All the deep reflexes on the left side seemed to be decreased a little, those on the right side increased. The white blood count was 25,000 at that time. A lumbar puncture gave grossly bloody fluid under an initial pressure of only 145. He was admitted with a diagnosis of subarachnoid hemorrhage.

In the course of a month in the hospital he improved fairly rapidly and regained most of his functions. The blood pressure was normal at entry and remained normal, 120/80, all the way through, the pulse about 100. There was Babinski on the left. Subsequent lumbar punctures on the fourth, seventh and twelfth days after entry all showed decreasingly bloody fluid and increasing xanthochromia. On discharge he was able to get about a bit, but his left side was still somewhat spastic. There were increased reflexes on the left and clonus of the left ankle.

History of interval. He had evidence of slight hemiplegia through the sixteen months' interval before he reëntered. He also complained of transient attacks of right lower quadrant pain.

Two nights before his second admission his mother noticed that he tossed about restlessly in his sleep. The following morning he ate very little breakfast and complained of severe right lower quadrant pain. Soon after breakfast he vomited. He was rather listless all the morning, complaining of right lower quadrant pain. At noon he vomited again. A doctor was called late that afternoon and gave powders which seemed to relieve the pain temporarily. An hour after taking the powders the patient vomited for the third time. He was put to bed, still complaining of pain in the right lower quadrant. In the evening he began to have diarrhea, first brownish stools, then greenish, finally merely mucus. He continued to have diarrhea all that night. At midnight he was unresponsive to questions and could not be aroused. The morning of admission he could be aroused, responded very slowly to questions, and still complained of right lower quadrant pain.

Second admission. He was brought to the Emergency Ward comatose at eight o'clock in the morning. He lay tossing in bed and not responding to questions. When he did lie quietly the right leg was drawn up. On examination the left hemiplegia still persisted. He had increased deep reflexes on the left and absent abdominal reflexes. The pupils were equal, regular and reacted normally. There was no choking of the disks so far as could be made out. It was very difficult to examine him because he was tossing about so much. The heart rate was 140 or 150. The sounds were of only fair quality. It was difficult to make out anything abnormal. The abdomen was rather soft, but whenever we pressed in the right lower quadrant he would groan and draw up his right leg.

He was seen in the Emergency Ward by a number of consultants. It was difficult to arrive at any conclusions. The surgeons thought he probably did not have a surgical abdomen and that even if he did have there was nothing to be gained by operation. He was seen by neurologists. Dr. Ayer thought he had a ruptured appendix and that there was nothing in the central nervous system to account for the present symptoms. An ear consultation was requested to make sure that the ears were normal and that there was no possibility that the condition might be a manifestation of brain abscess. Nothing abnormal was found.

The urine was loaded with hyalin and granular casts. The white blood count was 46,000.

*Recently senior interne on the West Medical service.

The temperature was 105°, the pulse 150. An x-ray film of the chest was perfectly normal. A lumbar puncture was negative.

The diagnosis was a problem. The boy was simply allowed to remain propped up in bed. We thought it might possibly be a very virulent early pneumonia. That afternoon we gave intravenous glucose. After about 100 cubic centimeters of fluid had gone in he suddenly became very cyanotic and went into a tonic convulsion. His limbs became spastic, his eyes seemed to stand out of his head and his breathing was stertorous. As soon as the convulsion started the intravenous infusion was stopped. He remained in that tonic condition, absolutely rigid, for half to three-quarters of an hour, then died.

Clinical Discussion

Dr. Gerald Blake: This boy was desperately ill when I saw him. The first question was whether there was any local infectious process. The abdomen was not typically board-like when we examined him. It was softer on the right than on the left. We felt that whether he had general peritonitis or not he was in no condition to be operated upon. The question came up whether this was a manifestation of septicemia from some focus that we could not find on examination. There was also a possibility of pneumonia that was not obvious at examination. Aside from a little stiffness of the neck and a Kernig which was not marked, this condition of the abdomen and the fact that he was very ill, we had no localizing signs. My feeling was that if he did not have general peritonitis he had a septicemia. There was a possibility of pneumonia. I could not make a more definite diagnosis than that.

Dr. A. William Reggio: We saw this boy with a question of surgical interference. We were not at all certain that he had general peritonitis, and if he did have it we thought it was too late to do anything. The only thing we could have done would have been to open the abdomen under local anesthesia and put in some wicks, and that would really have got him nowhere. In fact it would probably have hastened things for him. There was not the board-like abdomen; in fact the abdomen was almost soft. The temperature was 105°. He was an extremely ill boy. We felt that the only way of giving him any chance at all was by letting him alone. In these acute peritonitis conditions we can do nothing better than to let the patient alone.

Dr. Charles S. Kubik: What was the spinal fluid pressure when he came in the first time?

Dr. Kranes: It was 145. Of subsequent pressures the highest was 260.

Dr. Kubik: Did he ever get so that he could use his hand and fingers?

Dr. Richard S. Hawkes: At the time he returned to the hospital for the second admission his father told us he had as a residual only a little weakness of the left foot. We did some very close questioning at the time of the first admission as to the possible etiology of the hemorrhage. Nothing was ever found. We particularly inquired about lues in the family. There was no evidence of it.

Dr. Wyman Richardson: Subarachnoid hemorrhage is sometimes caused by an embolus from an endocarditis. I should think this might be considered in this case.

Clinical Diagnoses (From Hospital Record)

First Admission

Subarachnoid hemorrhage.

Second Admission

Brain abscess?
Ruptured appendix with peritonitis?
Pneumonia?
Dysentery (bacillary?).
Mesenteric thrombosis?

Anatomic Diagnoses

Acute appendicitis and periappendicitis.
General peritonitis, streptococcus hemolyticus.
Septicemia, streptococcus hemolyticus.
Varix of brain, probably congenital.
Pulmonary edema and congestion.
Hyperplasia and chronic inflammation of the thymus and mesenteric glands.
Hyperplasia of the bone marrow.
Acute tracheitis and bronchitis.
Acute terminal endocarditis, moderate.

Pathologic Discussion

Dr. Tracy B. Mallory: Autopsy showed as far as the body was concerned a diffuse peritonitis and an appendix which was small, thickened, a little firmer and a little more edematous than normal, but not ruptured. In gross we were not absolutely certain that the diffuse inflammatory process had its origin in the appendix. From the microscopic evidence, however, I think there is no question that the appendix was the primary source of the peritonitis.

There was nothing else of any essential importance outside the central nervous system. I am going to ask Dr. Kubik to tell you his findings there.

Dr. Kubik: At autopsy a large thin-walled vessel distended with blood was found in the upper Rolandic region of the right cerebral hemisphere. It was 3 to 4 millimeters in diameter and about one centimeter presented on the surface, the rest being buried in the substance of the brain. The brain in this region was considerably softer than normal. There was no discoloration or other evidence of hemorrhage.

After fixation of the brain in 10 per cent

formalin, coronal section disclosed a collection of thin-walled blood vessels in the upper part of the right anterior central convolution. They formed a longitudinal mass 0.5 centimeter in diameter extending from the surface to a distance of about 2 centimeters. The vessels ranged from 3 to 4 millimeters to less than one millimeter in diameter. Mesially there was an old softening stained with hemosiderin and partly organized, 4 centimeters in length and about 0.5 centimeter in diameter, running parallel to the mesial surface of the hemisphere and extending from the cortex of the upper Rolandic region to the corpus callosum. Laterally to the mass of the blood vessels was another organized softening about one centimeter in diameter. There was little if any distortion of the ventricles or the cerebral hemispheres.

Microscopically the lesion of the brain consists of a compact group of thin-walled blood vessels. Some of the vessels have irregularly thickened walls. The thickening consists of connective tissue with a laminated structure, usually outside the vessel, but in at least one case apparently within the lumen. The adjacent brain tissue is degenerated and replaced by a loose network of connective tissue and small blood vessels, and in some places by fibrous glia.

In spite of the fact that a streptococcus was recovered from the spinal fluid, there was no inflammatory exudate in the subarachnoid space.

In Weigert sections of the spinal cord there is a surprising degree of degeneration of the corresponding corticospinal tract considering the small size of the cerebral lesion and the fact that the hemiparesis had improved to such an extent.

The pathology found in the brain unquestionably accounts for the subarachnoid hemorrhage which occurred several months before, and presumably has no connection with the abdominal condition.

The lesion does not have the appearance of a tumor in the sense of a neoplasm. There is no distortion of the cerebral hemispheres to indicate displacement of tissue by an actively growing lesion. It is probably to be regarded as a congenital anomaly, something in the nature of a varix.

Our experience with so-called spontaneous subarachnoid hemorrhage indicates that it may be due to widely different causes and still give rise to the usual clinical manifestations. Some cases are the result of small, presumably congenital aneurysms. In a case with repeated attacks a frontal glioma was eventually found at operation. Not long ago in a patient who died several months after recovery from a subarachnoid hemorrhage postmortem examination disclosed arteriosclerosis with several vascular lesions of the brain, one of which extended to the subarachnoid space. In another case the only thing found was a diffuse hemorrhagic lesion situated in the tentorium. The cause of this lesion was not ascertained.

DR. MALLORY: I should like to emphasize the unusual character of this group of immensely dilated veins which were found in the arachnoid and running down a long distance into the brain tissue. Those veins are probably ten times the size of any normal vessels in that region. There is no positive evidence that we are dealing with a tumor; there is no evidence of any active proliferation in it; and I feel that Dr. Kubik is correct in considering it some form of congenital abnormality such as varix. I doubt if at any period since fetal life there had been any active growth. There is just a bare possibility that it may represent repair of some type of hemorrhage during birth. On the other hand it does not look like that. The vessels are so many and so large in diameter that it does not resemble any form of repair tissue I can recollect having seen. I think there is no question but that his first attack before admission consisted of rupture of one of those dilated veins, causing a small area of softening and then of course the secondary degeneration of the spinal cord Dr. Kubik spoke of as a still later event.

CASE 18382

UNUSUAL NEUROLOGICAL COMPLICATION IN A SURGICAL CASE

SURGICAL DEPARTMENT

PRESENTATION OF CASE

DR. HARRY M. SPENCE*: This case is somewhat like the first one, Case 18381, though this is a surgical case and is complicated by a neurological factor.

This is the case of a girl of twelve who entered the Emergency Ward complaining of pain in the right thigh of nine days' duration. She had fallen downstairs five days before the onset, injuring the outer aspect of her right knee, but had paid no more attention to it. Between the fourth and fifth days before she entered the hospital the region began to feel painful and tender.

The past history shows no mental aberration or birth trauma in the patient or any of her relatives.

Examination in the Emergency Ward showed a very robust young girl in a great deal of discomfort from an inflammatory process around the right knee, chiefly in the lower part of the right thigh. This was swollen, reddened and tender, particularly throughout the lateral aspect. The temperature was elevated, the white cell count 14,000. It was a fairly obvious case of osteomyelitis.

Under gas oxygen anesthesia a four-inch incision was made over the lower end of the femur

*Senior interne on the West Surgical service.

and 300 or 400 cubic centimeters of pus evacuated. A drill hole was made in the bone and pus was found in the marrow cavity.

During the next few days the patient continued to run a septic chart. Examination showed that she had a slight secondary accumulation of pus in the popliteal space, which was drained, again under gas oxygen.

The patient improved for the next few days, then again began to run a septic chart. X-ray showed a process involving the lower half of the shaft of the femur. Revision of the wounds with drainage was felt to be necessary. This was done, again under gas oxygen with preliminary morphia and atropin. At this operation everything went very well from the surgical point of view. The procedure took about thirty minutes. At the end of that time, as the dressing was being put on her leg, the patient was reported as being very blue and having ceased to breathe. On investigation it was found that the pulse, though fairly rapid, was of good quality, but the patient was not inspiring. Artificial respiration was begun immediately, oxygen and carbon dioxide being administered. After twenty minutes of artificial respiration the child resumed breathing at an irregular rate, taking a few gasps and then beginning again. She was sent back to the ward in this condition.

Three hours afterward the patient passed from a comatose into a maniacal state, entirely delirious. She shrieked out in a high pitched cerebral cry, inarticulate, tossing and flinging her arms about so that heavy doses of sedatives were required.

The child lived but thirteen days after this. Throughout this time the temperature was sustained at a high level, from 103° to 105°. The pulse was also elevated. The region of the operation drained fairly satisfactorily until about two days before death, when the knee joint itself became involved from above.

Clinical Discussion

Dr. Spence: We thought this child had a cerebral complication. A neurological consultant made a diagnosis of chorea insaniens with a toxic infectious basis, probably precipitated by the gas oxygen anesthesia.

Clinical diagnoses (from hospital record)

Osteomyelitis of the right femur.
Chorea insaniens.

Anatomic diagnoses

Osteomyelitis of the right femur. (Hemolytic streptococcus.)
Purulent arthritis of the right knee joint.
Degenerative lesions of the cerebral cortex and basal ganglia, probably ischemic in origin.

Pathologic Discussion

Dr. Tracy B. Mallory: At autopsy we found evidence of an extensive osteomyelitis and of extension of the infection into the epiphyseal line. There was complete separation of the shaft from the epiphysis and also purulent arthritis of the knee joint. Obviously the process in the knee joint was recent compared with the other, though it was impossible to tell how much more recent. We could not tell at what point the separation of the epiphyseal line had occurred. No x-rays were taken before the first operation. At the first examination after it, separation of the epiphysis was evident. We do not know whether it may have occurred before the first operation or not.

Besides this local sepsis there were small metastatic abscesses in the lungs and nothing else of much significance except the findings in the brain, which Dr. Kubik will demonstrate. It is a condition we have never seen before.

Dr. Charles S. Kubik: There were some changes to be seen in gross, intense congestion of the meninges and diffuse pinkish purple discoloration of the gray matter which did not seem to be due altogether to the congestion. No hemorrhages were found in it.

In material that has been fixed in ten per cent formalin and sectioned there is a well defined pale grayish zone from one to two millimeters in diameter in the deep layers of the cortical gray matter. It is found in practically all parts of the cerebral cortex of both hemispheres. Well-defined lesions of the basal ganglia are not apparent to the naked eye.

Microscopic examination. The vessels of the pia arachnoid are congested. The subarachnoid space contains numerous red blood cells and a few endothelial cells.

In the deep layers of the cortical gray matter and in the corpus striatum and the optic thalamus there are circumscribed lesions characterized by destruction of myelin, proliferation of glia elements and intense proliferation of blood vessels. Axis cylinders passing through the lesion are preserved but damaged. Ganglion cells are diminished in number and those that remain exhibit degenerative changes.

In the cerebral cortex the lesion consists of a more or less continuous well defined zone, up to two millimeters in width, with a smooth inner and an irregular outer margin, situated in the deep layers of the gray matter, the inner margin corresponding to the junction of the white and the gray matter. Within the lesion practically all myelin is destroyed. There is extensive proliferation of microglia with numerous granular corpuscles containing fatty material and particles of myelin. Many large mitotic cells are observed. Glia cells with large clear nuclei and well stained ameboid cell bodies, probably astrocytes, also appear to be increased in

number. While axis cylinders appear to be practically undiminished in number, many of them are swollen or have a beaded appearance and most of them exhibit some degree of alteration. Ganglion cells within the lesion are diminished in number. Most of those that remain appear to be severely damaged; some are very pale, somewhat shrunken or swollen and vacuolated and have pale nuclei; others are shrunken, more deeply stained, and have small deeply stained nuclei. Neurophagia is not observed. Outside the zone of severe degeneration ganglion cells are not normal; they are pale, no Nissl bodies are observed, and the cytoplasm has a homogeneous finely granular appearance. Some of them have shrunken, somewhat distorted and eccentric nuclei, which however have well defined nuclear membranes and are not deeply stained.

There is congestion of the cortical blood vessels. In the zone of degeneration the capillaries are greatly increased in number and there is extensive proliferation of their cellular elements. No hemorrhages are observed.

Lesions of the nature described are found in sections of cortex taken from various parts of the brain, and essentially the same findings are present in the corpus striatum and the optic thalamus. They are not observed in tissues consisting purely of white matter, nor are they found in sections of the pons, the cerebellum, the dentate nucleus or the spinal cord.

The pathology is in all probability due to anoxemia which was not severe enough or of sufficiently long duration to cause immediate death, but resulted in the lesions seen here. The proliferation of the capillaries and the glial changes are part of a reparative process, a reaction to the destruction of ganglion cells and myelin. They are not found in ischemic lesions occurring shortly before death. The severity of the process in the deep layers of the cortex and the circumscribed zone of myelin degeneration are striking and interesting features which would seem to indicate either that the blood supply of these parts is not so rich as it is in the outer layers of the cortex or that the tissues are more vulnerable.

Lesions very like these and with a similar distribution have been found in carbon monoxide poisoning, and it is more than likely that a similar mechanism operates in both cases.

DR. MALLORY: It is important to bear in mind that in spite of the diagnosis of chorea insaniens, the condition has of course no relation to Sydenham's chorea, which is an entirely different disease.

The New England
Journal of Medicine

SUCCESSOR TO
THE BOSTON MEDICAL AND SURGICAL JOURNAL

Established in 1828

Published by THE MASSACHUSETTS MEDICAL SOCIETY under the jurisdiction of the

COMMITTEE ON PUBLICATIONS

HOMER GAGE, M.D. JOHN W. BARTOL, M.D.
ROGER I. LEE, M.D. ROBERT B. OSGOOD, M.D.
EDWARD W. TAYLOR, M.D. (Deceased)

EDITORIAL STAFF

REID HUNT, M.D. STEPHEN RUSHMORE, M.D.
JOHN P. SUTHERLAND, M.D. HANS ZINSSER, M.D.
GEORGE R. MINOT, M.D. BENJAMIN WHITE, PH.D.
FRANK H. LAHEY, M.D. HENRY R. VIETS, M.D.
SHIELDS WARREN, M.D. ROBERT N. NYE, M.D.
GEORGE L. TOBEY, JR., M.D. ROBERT M. GREEN, M.D.
C. GUY LANE, M.D. CHARLES C. LUND, M.D.
WILLIAM A. ROGERS, M.D.

WALTER P. BOWERS, M.D., *Managing Editor*

ASSOCIATE EDITORS

GEORGE G. SMITH, M.D. WILLIAM B. BREED, M.D.
JOSEPH GARLAND, M.D.

THE NEW HAMPSHIRE MEDICAL SOCIETY
PUBLICATION COMMITTEE

D. E. SULLIVAN, M.D. H. H. AMSDEN, M.D.
R. H. MARCOTTE, M.D.

THE VERMONT STATE MEDICAL SOCIETY
PUBLICATION COMMITTEE

WILLIAM G. RICKER, M.D. C. F. DALTON, M.D.
J. A. WARK, M.D.

SUBSCRIPTION TERMS: *$6.00 per year in advance, postage paid for the United States, Canada $7.04 per year, $8.52 per year for all foreign countries belonging to the Postal Union.*

Material for early publication should be received not later than noon on Saturday. Orders for reprints must be sent to the Journal office, 8 The Fenway.

The Journal does not hold itself responsible for statements made by any contributor.

Communications should be addressed to The New England Journal of Medicine, 8 The Fenway, Boston, Mass.

THE MEDICAL TRUST

Is there not an opportunity for some young researcher in science to do an "arbeit", and at the same time render a service to medicine and society, by clarifying the situation which involves the Medical Trust? There is needed a dispassionate description of the Trust, its nature, its powers, its activities. While many physicians have heard of the Trust, few if any whose testimony is trustworthy have come across its trail. It is like the ghost of Hamlet's father: " 'Tis here! 'Tis here! 'Tis gone!"

On certain occasions, in certain places, for example some of the hearings on medical matters in the State House, awareness of the existence of this Frankenstein's monster is detectable, if one is to believe what is said; but if one asks (with apologies to Browning and to Shelley)

"Ah, did you once see Trustie plain,
And did he stop and speak to you,
And did you speak to him again?
How strange it seems and new,"

the reply is that of Kipling's Tomlinson, for whom there was no place in Heaven or in Hell.

"Oh, this have I felt, and this have I guessed,
and this have I heard men say;
And this they wrote that another man wrote of a
carl of Norroway.

* * * * * * *

Nay, this I ha' heard", quo' Tomlinson, "and
this was noised abroad,
And this I ha' got from a Belgian book on the
word of a dead French lord."

Most physicians have come to regard the Medical Trust as non-existent in reality, as a figment of the imagination of incompetents in medicine who are annoyed that anyone should think of thwarting their desire to take advantage of the ill and the weak and the credulous and the ignorant, and of effecting a restriction of what they consider to be their right to provide something for suckers to suck.

Perhaps it is not the man of science with his microscopic eye that is needed, but a new Cervantes to tell again the story of the eternal Don Quixote with his new windmill.

STOVARSOL IN THE TREATMENT OF SYPHILIS

STOVARSOL, as Fourneau has named it, or acetarsone, has recently been heralded with considerable enthusiasm for its value in the peroral treatment of syphilis, particularly congenital syphilis in children[1, 2, 3]. Stovarsol, or 3-amino-acetyl-4-hydroxyphenyl-arsenic acid was 594 in Ehrlich's series of investigations which led to the discovery of "606" or arsphenamine. It was found to be unsuitable for intravenous usage by Ehrlich because it produced harmful results when injected under the skin of the mouse. Stovarsol contains 27.1 to 27.4 per cent of arsenic; neoarsphenamine contains 20 per cent and arsphenamine 32 per cent. It is now being manufactured in 0.25 gm. tablets; its ingestion is followed by the rapid excretion of arsenic in the urine, and, when given by mouth, is of extremely low toxicity.

A safe dosage for a nine weeks' course of treatment has been given as 0.005 gm. per kilo daily for the first week, 0.01 gm. for the second week, 0.015 gm. for the third week and 0.02 gm. for the fourth week, which is the maximum dosage used.

Few untoward reactions have been encountered; mild reactions consist of slight elevations of temperature, and diarrhea. Transient exanthems have been noted, and also arsenical dermatitis. One of Maxwell and Glaser's ten cases died with flaccid paralysis.

On the other hand, the positive Wassermann test is commonly reversed after one to two courses of treatment, and the lesions tend to clear rapidly. Marked beneficial effect on the

general physical condition of infants and children was also noted. The opinion seems to be that stovarsol has a definite place in the treatment of congenital syphilis.

REFERENCES

1 Maxwell, C. H., Jr., and Glaser, Jerome: Am. J. Dis. Child., 43: 1461, June, 1932.
2 Rosenbaum, Harold A.: Am. J. Dis. Child., 44: 25, July, 1932.
3 Abt, Arthur F., and Traisman, Alfred S.: Jour. of Pediatrics, 1: 172, August, 1932.

THIS WEEK'S ISSUE

CONTAINS articles by the following named authors:

ERNSTENE, A. CARLTON. A.B., M.D. State University of Iowa 1925. Interne, Henry Ford Hospital 1925-1926. Assistant Resident Physician, Thorndike Memorial Laboratory, Boston City Hospital 1926-1927. Resident Physician, Thorndike Memorial Laboratory, Boston City Hospital 1927-1928. Assistant in Medicine, Beth Israel Hospital 1928-1932. Assistant in Medicine, Harvard Medical School 1927-1930. Faculty Instructor in Medicine, Harvard Medical School 1931-1932. Cardiologist, Cleveland Clinic, Cleveland 1932-. His subject is "The Anatomy and Physiology of Congenital Cardiovascular Disease." Page 523. Address: Cleveland Clinic, Cleveland, Ohio.

SPRAGUE, HOWARD B. A.B., M.D. Harvard University Medical School 1922. Assistant Physician, Massachusetts General Hospital. Visiting Physician and Research Director, House of the Good Samaritan. Assistant in Medicine, Harvard Medical School Courses for Graduates. His subject is "Congenital Heart Disease. Clinical Types and Diagnosis." Page 525. Address: 270 Commonwealth Ave., Boston.

JONES, T. DUCKETT. B.A., M.D. University of Virginia Medical School 1923. Instructor in Medicine, Harvard Medical School. Research Director, House of the Good Samaritan. Assistant in Medicine, Massachusetts General Hospital. His subject is "Rheumatic Fever." Page 529. Address: 25 Binney St., Boston.

BREED, WILLIAM B. M.D. Harvard University Medical School 1920. Associate Physician, Massachusetts General Hospital. Visiting Physician, House of the Good Samaritan. His subject is "Diagnosis and Clinical Signs of Rheumatic Heart Disease in Children." Page 530. Address: 264 Beacon St., Boston.

ROBEY, WILLIAM H. M.D. Harvard University Medical School 1895. Clinical Professor of Medicine, Harvard University. Chief of the Second Medical Service, Boston City Hospital. Consulting Physician, Milton and Norwood Hospitals. His subject is "Treatment of Rheumatic Heart Disease." Page 533. Address: 202 Commonwealth Ave., Boston.

DANA, HAROLD WARD. A.B., M.D. Harvard University Medical School 1905. F.A.C.P. Visiting Physician, Boston City Hospital. Associate Professor of Medicine, Tufts College Medical School. His subject is "Sources of Error in Blood Pressure Readings." Page 535. Address: 429 Beacon St., Boston.

BREMER, J. L. A.B., M.D. Harvard University Medical School 1901. Hersey Professor of Anatomy, Harvard Medical School. His subject is "Hypospadias and Epispadias: a Philological Note." Page 537. Address: Harvard Medical School, Boston.

BURBANK, REGINALD. B.A., M.D. Cornell University Medical College 1915. Assistant Surgeon, New York Orthopaedic Hospital, 1916-1925. Chief of Arthritis Clinic, Cornell University Medical College 1916-1918, and Bellevue Hospital 1918-1926. Instructor in Orthopaedics, Bellevue Hospital Medical College 1918-1926. Consultant on Arthritis, Brooklyn Hospital. His subject is "The Etiology and Treatment of Chronic Arthritis." Page 540. Address: 6 East 78th St., New York City.

GLENN, FRANK. M.D. Washington University 1927. Staff of the New York Hospital, New York City. His subject is "Pilonidal Sinus." Page 544. Address: New York Hospital, New York City.

MISCELLANY

A RADIO MESSAGE PREPARED AND SPONSORED BY THE COMMITTEE ON PUBLIC EDUCATION OF THE MASSACHUSETTS MEDICAL SOCIETY FOR THE DEPARTMENT OF PUBLIC HEALTH

COMMUNITY PUBLIC HEALTH*

BY PROFESSOR C. M. HILLIARD

Viewed from the standpoint of real values to mankind, there can be no doubt that the most important contributions of modern science during the last half century have been made in the field of public health. It is vastly more important to have conquered diphtheria by immunization than to have conquered the air with the airplane; it is of greater economic importance to have cut the death-rate from tuberculosis to one-quarter of its former rate than to have built the Panama Canal. More human happiness results from the fact that from 350,000 to 400,000 people escape from the deadly, or debilitating, attacks of typhoid fever than has resulted from the introduction of the radio, or cinemas.

Although statesmen have long recognized the importance of protection of the health of the people as an essential function of government, still the establishment of distinct administrative units to serve this purpose is of relatively recent origin.

*Broadcast: June 21, 1932. Time: 4:20 P.M. Place: Hotel Bradford, Boston.

The first local Boards of Health in America were established in Massachusetts in 1797, and the Massachusetts State Board of Health was not created until 1869. Even today there are thousands of communities in America without anything approaching adequate public health service. In our largest cities the quality of health service offered is very unequal, as is revealed by the per capita annual expenditures by one hundred of the largest cities. The largest per capita amount spent in 1929 by any city was 153.3 cents and the least was 30.1 cents. It is at least gratifying to learn that since 1923 the health expenditures for the group of cities studied has increased from an average of 59.1 cents to 73.2 cents or 23.9%.

The need for health work, and the obligation to do such work, becomes greater as our knowledge of the science of public hygiene increases. The futility of such measures as fumigation, house quarantine, rubbish and garbage disposal, and most nuisance abatement, which dominated public health practices up to within times that our fathers, or even we ourselves, can remember, did not perhaps justify great expenditures, or the employment of highly trained personnel. The work was largely that of a sanitary police officer, but with the dawn of the era of modern bacteriology all this was changed. The nature of disease microbes, their specific sources, and the ways in which they spread from person to person, and the science of immunity, and its practical application, gave definite tangible weapons of defense and combat against disease into man's hands. He could, for the first time, prevent certain diseases which had hitherto been absolutely uncontrollable. Mystery, superstition, and fear were replaced by knowledge and confidence. There are things that could be done, and should be done.

More recently the importance of health habits and health attitudes in people generally has come to be recognized as of great significance. Emphasis is placed more and more upon individualizing health work; that is, bringing the trained and the lay persons together for health educational work, for consultation and medical examination at the clinic or health centre, and in other ways promoting health in its positive aspects, rather than merely preventing disease. The public health nurse has become one of the most important members of health departments, and it is estimated that 40% or more of a health department's entire appropriation should be used for the employment of such persons.

The section on Health Service and Administration of the White House Conference which I have been asked to discuss has to do with the organization, and functions of agencies, both official and non-official, that definitely promote health and well-being, and prevent unnecessary illness and death among children.

For the most part, the matters considered in this section rebound to the benefit of people of all age groups, and not to children only. Community sanitation, clean, safe milk, and tuberculosis prevention work are of general importance, and if neglected in an hypothetical childless city, would quickly lead to health wreckage in the adult population.

On the other hand, the prevention of milk-borne tuberculosis of cow origin, which is transmissible to children only, diphtheria immunization programs, and the staffing of health departments with nurses doing infant and child hygiene work are obviously of specific value to the young.

This section would emphasize, therefore, the organization and services that will be of principal benefit to children, but cannot entirely divorce the discussion from the more general matters.

Organization is basic. Without personnel, properly trained for the special fields of public health activity, devoted to their work, and welded together under an executive head, we cannot hope for efficiency in health departments. We would not expect farmers to operate complicated textile machinery, or bank clerks to make good farmers, yet there has, curiously enough, been the impression that a taxicab driver may serve satisfactorily as a health officer, or a carpenter may be competent to serve on milk inspection.

Official health work in the United States, like other governmental organizations, is decentralized. The Federal work is somewhat diffuse, being scattered through different departments, but the United States Public Health Service is the most important unit.

Massachusetts has a highly organized and efficient Public Health Department with eight distinct divisions, including Divisions of Hygiene, Tuberculosis, Sanitation, and Communicable Diseases. The cities and towns in the State are required to provide Boards of Health; what their composition and work may be is largely up to each community. Our towns and small rural communities are often woefully lacking in anything that resembles adequate personnel and health work. Issuing permits to garbage collectors and to milk dealers, and tacking cards on doors of homes where there are cases of chicken pox or measles constitute the chief, if not the only, activities.

Citizens demand, and willingly pay for, fire protection and police protection; we still have fence viewers and inspectors of slaughter, showing the sound sense of our ancestors, but also the inertia of the government of today. But of health and life, our own and our children's, we are careless, often not applying to their fullest extent the most remarkable and beneficent discoveries of all ages that save life and extend the life span.

The first, and most important recommendation is then a full-time, properly trained, health personnel. Where communities don't have full-time jobs to offer, or money enough to pay for this work, they may combine with nearby communities to employ a health officer, milk and sanitary inspector, community nurse, or whatsoever trained persons may be necessary to give at least minimum adequate

health service. Recognition is given to the extremely valuable services of private agencies working in the public health field. They have often pioneered in such fields as community nursing, and infant welfare. They have, and always will have, a distinct contribution to make to health work. It is urged that the work of such organizations be closely and understandingly allied to that of official health bodies for mutual aid to achieve optimum results.

A second report of this section deals with milk, unquestionably our most universally used and most valuable food, especially in relation to child nutrition. Milk is also, potentially, man's most dangerous food from the standpoint of the spread of diseases. From the cow may come tuberculosis, undulant fever, or septic sore throat, while man, through handling, may seed milk with scarlet fever, diphtheria, and typhoid germs. Besides this, milk on account of its origin, handling, and fluid condition may pick up more microbes than any other food. We do not need to argue the desirability of producing milk from healthy cattle only. Barnyard manure or other dirt in milk doesn't appeal to any one. All will agree, then, that the cleanest milk that it is reasonable to demand, is milk produced from healthy cows, especially cows free from tuberculosis. The health specialist is not satisfied with this alone, but realizes that most, if not all milk to be safe, must be heated. The panacea against milk-borne disease is pasteurization; a scientific commercial heating of milk under rigid control and supervision, to a temperature, and for a time that certainly kills every disease microbe that can lurk in milk, but which does not impair its food value. The Committee, then, recommends milk produced from tuberculosis-free cows in clean dairies, clean handling, and pasteurization, all of this checked by proper milk inspection.

The third and last subdivision of this section deals with communicable disease control. Most of the epidemic diseases occur chiefly in childhood, and are especially fatal in the early years of life. The public holds too lightly the consequences of those two ubiquitous diseases, measles and whooping cough. Familiarity ever breeds contempt. These two diseases today are the most frequent causes of death in children under five years old from infectious agents. It is supremely important for parents to realize this, and to protect little children from these diseases, or to procure medical advice if infection occurs. Scarlet fever and diphtheria are duly respected as they should be, though the former disease causes less than one-third the fatalities of whooping cough.

Every Board of Health should adopt and enforce regulations which conform to the latest knowledge regarding the quarantine and isolation of persons ill with communicable diseases, and those exposed to contagion. In Massachusetts, a set of minimum requirements has been drawn up by a committee of experts, and should serve as a guide to every community in the State.

There are two diseases that could be rendered obsolete by the application of present-day knowledge. One of these, smallpox, needs no discussion, as the experience of 135 years bears its own testimonial. Where vaccination has been continuously and conscientiously practiced, this loathsome scourge has disappeared; where neglected it still attacks and maims, or kills many of its victims. The compulsory vaccination laws for school children should be adopted, upheld, and enforced.

The other disease referred to has become controllable only within the last dozen years. What has been accomplished with smallpox may now be done with diphtheria. Let us pray, since diphtheria is fatal chiefly in childhood, that the same shocking delay and neglect shown by people during the last century with regard to smallpox, will not prevail with this disease. We have the miracle of a weapon put into our hands with which to allay the terror and tragedy of this highly fatal disease! Any parent who fails to have his child protected is indifferent to a duty; and any community not providing the facilities for diphtheria immunization would seem to be criminally negligent. Children should be protected as early as possible; immunity probably lasts for life.

Tuberculosis, year in and year out, remains man's greatest foe. We have no single way of combating the "white plague", and yet by attacks upon it from various angles, it has yielded steadily until now it causes scarcely one-third the ratio of deaths to population that it caused a half-century ago. The contest against tuberculosis has been pressed back earlier and earlier in childhood until now it is realized that sound health building in this period, and avoidance of infection early in life are among the most essential preventive measures. The early discovery of tuberculosis, or better, the discovery of the predisposition to this disease, so that treatment and resistance can be built up, are most important factors.

These are but a few of the most salient of the findings and recommendations of that great body of experts that worked on these problems of Health Service and Administration at the request of President Hoover. If the communities and the people all over the land would but follow the recommendations, what a lot of unnecessary suffering would be prevented! How many lives would be saved! How much happier and healthier our children would be! There is a challenge in the knowledge of how to achieve these results.

THE MORTALITY RATE DUE TO AUTOMOBILE ACCIDENTS

Statistics based on the records of deaths due to automobile accidents recorded in eighty-six large cities of the United States, compiled by the Bureau of the Census, shows that the peak is reached in January, drops rapidly through February and March, shows a fairly stable rate through the summer months averaging about eighteen or nineteen per

one hundred thousand persons and after September rises quite continuously up to the first of the year, the highest rate in January, 1932, having been about thirty-one. The autumn rise has been explained by some as due to earlier twilight and darkness and worse road conditions which are not taken into consideration by operators, hurrying toward home at the end of the day's work, wet leaves and frost on the roadway which promote skidding, all of which seem to be factors in the rising accident tide. Another explanation may be that a new group of operators whose experience has been confined to the better road conditions in summer may not have acquired caution.

The problem is not confined to the mortality rate but extends to large numbers of injured persons who, although escaping with their lives, are subjected to suffering and crippling disabilities which may last throughout life.

Until the irreducible minimum of these accidents shall have been reached, the great burden thus imposed calls for more preventive measures than have been provided. It may be necessary to have greater care exercised by the authorities in determining the physical conditions of applicants for licenses as well as in the imposition of penalties.

Although many safeguards are provided by traffic signals and proficient officers, the dangers are so evident that more study of the possibilities of diminishing the hazards of automobile travel seems to be in order.

MORTALITY RATES

Telegraphic returns as compiled by the Bureau of the Census from 85 cities with a total population of thirty-seven million for the week ending Sept. 3, indicate a mortality rate of 9.9 as against a rate of 9.8 for the corresponding week of last year. The highest rate (19.3) appears for Washington, D. C., and the lowest (4.1) for Waterbury, Conn. The highest infant mortality rate (15.7) appears for Lowell, Mass., and the lowest for Canton, Ohio, Long Beach, Calif., Lynn, Mass., New Haven, Conn., Schenectady, N. Y., and Tacoma, Wash., which reported no infant mortality.

The annual rate for 85 cities is 11.4 for the thirty-five weeks of 1932, as against a rate of 12.2 for the corresponding period of the previous year.

PROPORTION OF CHILDREN PROTECTED AGAINST DIPHTHERIA

EXCERPTS FROM THE N. Y. CITY HEALTH DEPARTMENT BULLETIN, AUGUST 20, 1932.

The very excellent studies made by Godfrey of the influence of diphtheria immunizations on diphtheria mortality indicate that at least 30 per cent of young children must be immunized in order to effect a significant reduction in the diphtheria death rate. The death toll of diphtheria is especially high in the first few years of life, for which reason the Department of Health is directing its efforts principally to the immunization of preschool children. At the present time . . . about 36 per cent of the child population under six years of age has been immunized against diphtheria. . . .

Private physicians, it may be noted, perform about one-quarter of all the immunizations. . . .

We find that at present, out of an estimated population under six of 635,000, the number of immunized children is 229,355, or 36 per cent. That the immunizations have been effective in reducing diphtheria mortality in New York City has been shown by statistical analyses in previous issues of the *Weekly Bulletin*. The work must, of course, be continued, and we hope the time will soon come when all parents will have their infants immunized before their first birthday.

RECENT DEATHS

FAULKNER — WILLIAM EDWARD FAULKNER, A.B., M.D., of Keene, N. H., died in that city September 16, 1932. He was a graduate of Harvard University in the class of 1887 and of the Harvard Medical School in 1890 subsequently studying in Vienna and Berlin. Dr. Faulkner was a visiting surgeon of the Boston City Hospital for twenty years retiring in 1922. He was commissioned a Lieutenant Colonel of the British Army in the World War and later commissioned as Major in the American Medical Corps.

After removing to Keene he was made consulting surgeon at the Elliot Community Hospital. He is survived by a widow, four children, two brothers and a sister. He retained membership in the Massachusetts Medical Society to the time of his death.

FILLEBROWN — CHARLES DALTON FILLEBROWN, M.D., aged 70 years, formerly of Boston, Mass., died in Camden, Maine, September 16, 1932.

Dr. Fillebrown was the son of Dr. Thomas Fillebrown, formerly of Boston and Plympton, who died in 1908.

Dr. Charles D. Fillebrown practiced as a nose and throat specialist for over thirty years in Boston having an office on Boylston Street. He was born in Auburn, Maine, graduated from Exeter Academy, Exeter, N. H., and received his M.D. degree from the Harvard University Medical School. He was formerly a Fellow of the Massachusetts Medical Society terminating his membership in 1901. He is survived by a widow Mrs. Louise Fillebrown of Camden, Maine, and one daughter Mrs. Robert Wetmore of Boston.

HALLETT—MR. G. WEBSTER HALLETT, a prominent figure in health matters in Barnstable County, died at his home in Osterville on September 14. He was born at Marston's Mills, one of the villages of the town of Barnstable, some seventy-two years ago, went to sea while a mere boy, and in later years has been in the plumbing business in Osterville. As a practical sanitary expert, he has been well known

in a great many sections of this State, as a speaker and an adviser. For about twenty years he had been active in local health work. For eighteen years he was a member of the board of health of Barnstable, was active in the formation of the Southeastern Massachusetts Association of Boards of Health, of which he was secretary for sixteen years, and two years, its president, resigning in 1931. He was likewise a most important factor in the establishment of district health control in the Barnstable County Health Unit, the first of its kind in this state. This was the direct outcome of another project in which Mr. Hallett was keenly interested, the Cape Cod Health Bureau Association, organized some eleven years ago, and of which Mr. Hallett was a president. This association continues its good work today, supplementing the official accomplishments of the Unit by such aid as a voluntary society is so often able to afford.

Mr. Hallett was a director of the Cape Cod Hospital, a trustee of the Barnstable County Sanatorium, and outside of his home district, an active and influential member of the Massachusetts Association of Boards of Health. He remained in close touch with these organizations as well as with his business and home town interests till the time of his death. He leaves an enviable record.

NOTICES

CLINICS IN MEDICINE AT PETER BENT BRIGHAM HOSPITAL

On Thursdays at 3:30 P.M. in the Amphitheatre of the Peter Bent Brigham Hospital Dr. Henry A. Christian, Hersey Professor of the Theory and Practice of Physic, Harvard, and Physician-in-Chief, Peter Bent Brigham Hospital, will give a series of clinics which members of the medical profession are cordially invited to attend. The first clinic will be given on October 6, 1932.

Also physicians and medical students are invited to attend ward rounds of the Medical Staff of the Peter Bent Brigham Hospital held from 10-12 o'clock on Saturdays.

AMERICAN BOARD OF OBSTETRICS AND GYNECOLOGY

ANNOUNCEMENT OF COMING EXAMINATIONS

The next written examination of the American Board of Obstetrics and Gynecology will be held on Saturday, October 22, at 2 p. m., in 19 different cities of the United States and Canada. In order to reduce traveling expenses for candidates special arrangement may be made through the Secretary for taking the written examination at any city other than those regularly specified where there is a Diplomate who can be empowered to conduct the examination. This arrangement does not apply to the general, clinical examination.

The next general, oral and clinical examination is to be held in conjunction with the meeting of the Pacific Coast Society of Obstetrics and Gynecology at Los Angeles, California, on December 7, provided there are sufficient applicants.

The only reduction in railroad fares that will be available will be possible seasonal or round trip special rates. Information regarding this should be obtained from any railroad agent. The candidates will have the opportunity also of attending the scientific sessions of this Society, as the examination is scheduled for the day preceding the opening of the session.

Applications for these examinations should be filed immediately. Lists close for Group B applicants on October 5, and for Group A applicants on November 15.

For application blanks and other information, address Dr. Paul Titus, Secretary, 1015 Highland Building, Pittsburgh, Pennsylvania.

REMOVAL OF OFFICES

Fletcher H. Colby, M.D., has moved his office from 6 Commonwealth Avenue, Boston, to 205 Beacon Street, Boston.

John Bryant, M.D., announces the removal of his office to 311 Beacon Street, Boston.

NOTICE OF MEETING

THE NEW ENGLAND DERMATOLOGICAL SOCIETY

The regular quarterly meeting of the New England Dermatological Society will be held on Wednesday, October 19, 1932, at the Rhode Island Hospital, Providence. There will be a clinical meeting at the Rhode Island Hospital at three o'clock and a buffet supper at the John M. Peters House at six o'clock.

WM. P. BOARDMAN, *Secretary.*

SOCIETY MEETINGS
CONGRESSES AND CONFERENCES

September 26, 27, 28—Eighth Clinical Congress of the Connecticut State Medical Society. See page 331, issue of August 18.

September 30, October 1—Scientific Program for Annual Meeting of the New England Surgical Society. See page 333, issue of August 18.

October 6—Clinic in Medicine at Peter Bent Brigham Hospital. See notice elsewhere on this page.

October 12, 13 and 14—American Academy of Physical Therapy. See notice on page 384, issue of August 25.

October 17-21—Clinical Congress of the American College of Surgeons. See page 1330, issue of June 23.

October 17-28—The 1932 Graduate Fortnight of the New York Academy of Medicine. See page 1375, issue of June 30.

October 19—The New England Dermatological Society. See notice above.

October 19—The New England Physical Therapy Society. First meeting of the season. Detailed notice will appear in a later issue of the Journal.

October 22 and December 7—American Board of Obstetrics and Gynecology. Announcement of Coming Examinations. See notice elsewhere on this page.

October 22-24—Health Education Institute. See notice on page 47, issue of July 7.

October 24-27—American Public Health Association. See notice on page 1321, issue of December 31, 1931.

October 24-28—Inter-state Postgraduate Medical Association of North America. See page 39, issue of January 7.

MIDDLESEX EAST DISTRICT MEDICAL SOCIETY

The program of the Middlesex East District Medical Society for the coming year is as follows:

September 28 at Bear Hill, luncheon at 1:00 P.M.
November 9 at Winchester.
January 11 at Wakefield.
March 15 at Melrose.
May, at the Meadowbrook Club, Reading.

ALLAN R. CUNNINGHAM, Secretary.

BOOK REVIEWS

AN INSTRUCTIVE PAMPHLET

A very useful brochure setting forth the solution of the problems of feeding a family has come to the editorial desk under the title "How to Select Proper Food For The Family". This has been compiled by Dr. Stanley H. Osborn, Connecticut's Commissioner of Health, and if the advice contained therein is generally applied by parents there will be improvement in the health and growth of children.

Diets for early life up to sixteen years are tabulated with explanations of nutritional and vitamin needs. Advice for dietary needs during pregnancy and for the tuberculous is included, and the economic problems of food supplies are apparent.

Even some physicians could find a great deal of information in this publication and, if copies are available for the laity, good will come from the distribution of them.

Surgery, With Special Reference to Podiatry. By EDWARD ADAMS, M.D., Professor of Surgery, The First Institute of Podiatry of New York. Int. Journal of Surgery Co. New York, N. Y. 1932. Price $5.00.

The first six chapters of this book may be summarized as a dictionary of surgical terms and general information concerning surgical bacteriology, asepsis, antisepsis, inflammation, wounds and contusions, hemorrhage, etc. The remainder of the book is arranged under headings of lesions and diseases and therapeutic measures. In an attempt to cover the whole field of surgery, even with special reference to the foot, it is inevitable that in a volume of less than 450 pages of text, erroneous impressions should be given. One illustration will suffice. The subject of Sub-deltoid or Sub-acromial Bursitis is covered by four lines which read as follows: "Inflammation of the bursa under the deltoid muscle. In this condition there is a calcareous deposit in the bursa; operating to remove the same is the proper procedure." There are also many misstatements, such as, "The great majority of chronic joint diseases are tubercular in origin." Some of the surgical therapy even in relation to foot conditions is positively bad, such as the unqualified advice to amputate a hammer toe, a condition usually easily curable by a simple bone plastic operation.

The volume may be a worthy attempt to raise the standard of podiatry, but in its present form it is an inaccurate attempt and we cannot recommend it either to general practitioners or to podiatrists.

Manual of Clinical and Laboratory Technic. By HIRAM B. WEISS and RAPHAEL ISAACS. 4th ed., reset. Philadelphia and London: W. B. Saunders Co., 1932. Pp. 117. Price $1.50.

The data in the manual and the tests are compiled with the idea that the book may be used as a guide for the standardization and correlation of work, rather than as a textbook of clinical microscopy. It is planned to give the student or interne an outline which he can follow in the systematic study of his patients and as a handy reference book for the practitioner. The tests are those in most common use and only essential details are given, serving to recall the more comprehensive material studied in the classroom or derived from the larger textbooks on the subject. This book was formerly published by the Medical College Book Store of the University of Cincinnati College of Medicine and appeared first in 1922. The fourth edition has been undertaken by W. B. Saunders and Company and there has been a fairly complete revision, and rearrangement. The printing is clearer and the paper of a better grade. The contents include history and physical examination outline, directions for various laboratory examinations and technical procedures. As a sort of appendix there is a table of nutritive food values. There is an adequate index.

In the description of the technique for paracentesis, the authors have failed to state that before doing chest, peritoneal and lumbar punctures, the operator should scrub his hands and the instruments should be sterilized; otherwise there are very few criticisms to be made.

Papers on Surgery and Other Subjects. By GEORGE TULLY VAUGHAN, M.D., LL.D., F.A.C.S. Published by F. W. Roberts Company, Washington, D. C.

This is a volume of original articles by the author with an account of the problems incident to a busy surgeon with his methods of treatment, setting forth the standard procedures modified according to circumstances.

This book will be read with interest by the friends and associates of the author and also by others who find entertainment in following the experiences of a fellow craftsman.

Problèmes Actuels de Pathologie Médicale. Second Series. By A. CLERC and others. Masson et Cie. Paris, 1932. Pp. 337. Price 40 fr.

This second series of clinico-pathological treatises on various phases of medicine, edited by Professor Clerc, continues the interesting type of discussion presented in the first series. Three sections are devoted to various phases of renal disease. The two most interesting sections in this volume are the chapter on blood changes in radiologists, by Aubertin, a subject rather too much neglected in our medical literature, and a concise and very readable review of hyperglycemia by Laroche.